"CLINICAL PROFILE OF PATIENTS ADMITTED WITH RODENTICIDE POISONING"

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THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

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CERTIFICATE

This is to certify that this dissertation entitled "**DISSERTATION ON CLINICAL PROFILE OF PATIENTS ADMITTED WITH RODENTICIDE POISONING"** is the bonafide work of **Dr.GOVINDA MURUGAN .T** in partial fulfilment of the requirements for M.D Branch -I (General Medicine) Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in APRIL - 2013 under my guidance and supervision during the academic year January- 2012 to November - 2012.

Prof. Dr. P.G.SANKARANARAYANAN, M.D.,
Unit Chief M-II,
Department of Internal Medicine,
Thanjavur Medical College,
Thanjavur - 613 004.

Prof.Dr.S.MUTHUKUMARAN, M.D.,
Head of the Department,
Department of Internal Medicine,
Thanjavur Medical College,
Thanjavur - 613 004.

Prof. Dr. C.GUNASEKARAN, M.D.DCH,
DEAN, I/C
Thanjavur Medical College,
Thanjavur - 613 004.

DECLARATION

I, **Dr.GOVINDA MURUGAN.T**, solemnly declare that the dissertation titled **" CLINICAL PROFILE OF PATIENTS ADMITTED WITH RODENTICIDE POISONING "** is a bonafide work done by me at Thanjavur Medical College, Thanjavur during January 2012 to November 2012 under the guidance and supervision of **Prof. Dr. P.G.SANKARANARAYANAN, M.D.,** Unit Chief M-II, Thanjavur Medical College, Thanjavur.

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ABSTRACT

Background:

Rodenticide poisoning, the second most common poisoning in our area. Three types of rodenticides are available in our area, namely phosphorus compound (ratol paste), zinc phosphide(powder), superwarfarin(bait). Among these compounds, phosphorus causes death in patients. Since only limited studies are available about rodenticide poisoning in India, this study was done, about the clinical profile of patients admitted with this type of poisoning and correlating various parameters with the mortality.

Methods:

This was cross-sectional study done at Thanjavur medical college hospital, Thanjavur, conducted among 60 patients , admitted with alleged history of ingesting rodenticides in past 24 hours before admission, satisfying inclusion and exclusion criteria. Detailed history, examination with laboratory parameters such as serum bilirubin on admission, after 4^{th} day and afterwards, INR value, SGPT, serum creatinine was estimated and they are correlated with the mortality of the patients. The results being analysed with chi-square and ANOVA test.

Results:

In this study, mortality was 20%, all due to phosphorus compound. Most common age group of 20-30 years,mostly from poor socioeconomic status. 70% ingested phosphorus compound, 18.3% ingested phosphide and 11.7% ingested super warfarin compounds. Mortality was higher in delayed hospitalisation. Abdominal pain was the most common symptom and jaundice observed on 4^{th} day of admission after ingesting phosphorus.

No significant association with age, sex, socio-economic status, marital status, abdominal pain and bleeding manifestations. But there was significant correlation with quality of compound, time delay, jaundice, oliguria, serum bilirubin, SGPT and serum creatinine level with mortality.

Conclusion:

Mortality was common in phosphorus compound , who developed jaundice on 4^{th} day of admission following acute hepatotoxicity reflected by elevated bilirubin, SGPT values and hence considered as deadliest compound and can be banned , having lethal dose of less than 1mg/kg. Further early administration of N-acetyl cysteine is recommended.

Key words;

Rodenticides, yellow phosphorus compound, N-acetyl cysteine.

CONTENTS

INTRODUCTION:-

Poisoning is mostly preventable, commonly suicidal and rarely being accidental form of death in developing agrarian countries. In rural India, poisoning forms the major share of emergency health care and of about one – fourth to one – third of intensive care admissions.

Among poisoning, in our region, next to organo-phosphorus poisoning, rat killer or rodenticide poisoning remains the second most common ingested poison.

However as compared to organophosphorus poisoning, there are only few literatures available for rodenticide poisoning. Almost every system is affected in rodenticide poisoning and also there is no definite treatment guidelines available .Regarding it's incidence, mode of action and management, only few studies are available. Further there is no positive clinical trials available regarding phosphorus paste poisoning , which remains the deadliest among rodenticide poisons.

Rodenticides are commonly available in almost every household, to prevent their stored grains from rodents, which is found everywhere. Since it is easily available and also cheaper than other

pesticides in the markets. Because of its easy availability in every house , it is often taken with suicidal indent or ingested accidentally by children.

Rodenticide is available in three different forms in our area, as follows –

1) Powder form - zinc phosphide

2) Paste form - yellow phosphorus

3) Bait form - super warfarins

Among these rodenticides , phosphorus being the most commonly and also most lethal poison , especially after 3-4 days of ingestion , as liver injury sets in. Since there is no sufficient datas available regarding rodenticides , I prefer to perform this study regarding its clinical profile.

AIMS AND OBJECTIVES:-

1) To study the clinical profile of patients admitted with rodenticide poisoning and their course in the hospital.
2) To correlate various parameters with the mortality.

REVIEW OF LITERATURE

RODENTICIDE POISONING

There was competition between humans and rodents for food since past. Due to his super brain, human developed some agents to kill the rodents. Rodenticides are produced in such a way to kill the rodents , which damage the crops in field , stored grains in godowns, bite peoples and also capable of spreading deadly diseases such as plague[1,2].

It includes variety of chemicals with various actions, which may be organic or inorganic, may be mild or highly toxic, but they kill them in a cost effective manner. Now the problem arise, because humans either accidentally or intentionally ingest these chemicals.

In earlier days rodenticides, were mainly made from plant such as strychnine or inorganic chemicals as thallium and arsenic trioxide .Now, they are manufactured from synthetic organic compounds

Some agents such as thallium are as dangerous to human as rodents. However their use being restricted nowadays to lessen the risk of toxicity in humans especially. But still, they are regarded as toxic, if they are misused.

Rodenticide is a product, which is available in the market, use to kill rodents, mice and other small animals.

Perfect rodenticide is a one as suggested [1,3] , " that effectively kills rodents , but is not toxic to humans or non- rodent pet" , which is yet to be discovered or synthesized .

However, now, a large group of less than perfect rodenticides are available in markets, which differs from each other in chemical composition and mode of action and its toxicity to humans.

Rodenticides are classified [4] in several ways as

1.) Inorganic or organic compounds
2.) Animal selectivity
3.) Nature and onset of symptoms and
4.) LD 50 in rats

Of these compounds; zinc phosphide and phosphorus comes under highly toxic compound, while superwarfarin being a low toxic compound.

Since zinc phosphide is available in most diluted form in market, toxicity is comparatively less.

Let now see the three individual compound in detail.

PHOSPHORUS

It is derived from greek word PHOS:- meaning light ; PHORUS :- meaning bringing .

It was first identified in urine by chemist HENNING BRANT in hamburg in 1669. The early use of phosphorus was in 1720 in medical books. It was used for colic, tetanus, gout and apoplexy. It was popular as tonic aphrodisiac, impressing on looker of its phosphorescent and explosive nature. Inspite of its toxicity, it was found in British pharmacopedia till 1932.

White phosphorus gained importance, since it was used as main ingredients in lucifiers or in match industry. It was invented by chemist Charles sauria of paris in 1830. It was mainly used in match industry. After some years it was banned world wide as of using in striking pad. On the match box, and now red phosphorus has replaced it, in striking pad as well as match head.

White phosphorus is now used to manufacture, insecticide, fertilizer as phosphoric acid and homemade wirework [5] which contain about 10% phosphorus.

Phosphorus in 15th element in table, it exist in 3 forms

i) black phosphorus →non toxic, not ignite spontaneously

ii) red phosphorus →faily innocuous→ intermediate reactivity

iii) white phosphorus →highly reactive and dangerous compound.

White phosphorus in tetramer (D_4), water insoluble, a waxy paste. It emit pale green glow, due to low reactivity with oxygen. Impurities in white phosphorus make it as yellow phosphorus.

Phosphorus on combining with oxygen, gives Phosphorus oxide with liberation of light and dense smoke with Garlic odour, On dissolving with water, it produces phosphoric acid.

It is rapidly absorbed and distributed primarily in the liver, kidney, intestinal mucosa, epidermis, follicles and pancreas

Other tissues such as lung, myocardium, spleen and renal medulla of moderate distribution , brain fat and muscle of low distribution[7].

In rats, sub-cutaneous lethal dose in 10mg /kg[8] while in human studies suggest lethal dose as 50mg or 1mg/kg.[9]. It is highly lipid solubility, high absorption occur after skin or mucosal exposure.

CLINICAL MANIFESTASIONS:

Mortality due to ingestion, range from 20 to 50% [10,11]

Indicators of poor prognosis [12]

i) ingestion of more than 1mg/kg

ii) severe electrolyte disturbance

iii) mental status changes

prolongation of Q-T interval

iv) more than 10 fold raise in alanine amino transferase

v) severe coagulopathy

vi) peak liver enzymes reached within three days of ingestion

Old literature, describes course of poisoning in 3 stages [13]

i) lasts- hours to days after ingestion manifested as irritation of digestive tracts, may cause arrhythmias and same neurological manifestations.

ii)II stage: digestive symptoms resolve, lasting for few days

iii) patients develop hepatic, renal and cardiac toxicities, mostly causing death. If recovery occurs, it takes few weeks

However most death occurs as result of fulminant hepatic failure within first week. Some delayed death occurs between 5-8 days due to cardiotoxic. Few deaths only occurs,if patient survive beyond this stage.

HEPATIC:

It produces hepatotoxicity in dose dependent manner [14]. Amino transferase rises in half of ingested patients, mostly begin to rise within one day of exposure. Time of enzyme peak and level rise differ significantly between patient who survives and who dies.

Patients who dies have average peak of sixteen times of normal, reached in 2-3 days. In those who survives, alanine aminotransferase (SGOT) rises eight times normal, reaching peak in six days .

Coagulopathy in 20 % of patients [5]

Serum triglyceride level fall as toxicity develops, there may be also increase in serum and urinary ketones

It consumes oxygen in liver cells , is a protoplasmic poison which uncouples oxidative phosphorylation , in turns leads to decrease in intrahepatocyte ATP levels[8]

It also affects transition of triglyceride as beta-lipoproteins. Massive hepatic steatosis, hallmark of white phosphorus toxicity, rise in triglyceride level in two hours , peaking in two days . Hepatic necrosis is particularly in zone 1 , distinct from acetaminophen and carbon tetrachloride , hepatic glycogen are decreased due to increased glucose-6- phosphatase activity .

A non specific finding of huge increase in rough endoplasmic reticulum, is typically found in this poison [17,18]

Liver biopsy showed signs of acute hepatocellular necrosis also showing fibrosis and piecemeal necrosis

SKIN, MUCOSA AND GASTROINTESTINAL TRACT:-

Burns produced by this poison is extremely painful with characteristic garlic odour, yellow colour and necrotic base. it heals slowly like other chemical burns , and also require prolonged hospital stay[19,20] , which may be of second degree or third degree burns involving 15% of body surface area , may result in death[12] ,due to absorption of phosphorus .

Mucosal erosions were also noted, when it get exposed to mouth, eyes, it may show bullae , injection.

Thus following intake of phosphorus, it may shows oral burns, vomiting, loose stools, abdominal pain and even bleeding, as haematamesis. the vomitus and stool show typical garlic odour , and it also appear as glistening in dark room , hence it is also mentioned as SMOKING STOOL SYNDROME .[19]

The main mechanism being exothermic reaction . If it penetrates dermis, causes tissue damage, due to production of phosphoric acid, when reacting with oxygen. Digestive system is less involved than skin, probably due to low oxygen concentration. Severe mucosal involvement is also explained due to generation of phosphoric acid.

CARDIOVASCULAR SYSTEM :-

Early death within twenty four hours, after intake of phosphorus is mainly due to cardiovascular collapse

Electrocardiograph taken during interval hours of admission may reveal. QT prolongation, atrial fibrillation, ST depression, which may be due to electrolyte deficiency, mainly of calcium.[20,21]. Cardiac arrest also occurs, after few hours of even dermal exposure, it generally occurs 4-10 hours after absorption. Cardiac arrest without any rhythm disturbances or myocardial injury

are also reported. hypo calcemia also causes cardiomyopathy and mimics like myocardial infarction.[22,23]

Usually there are no morphological changes, however intestinal edema and vacuolated cytoplasm urine noted.

NERVOUS SYSTEM:

It produces neurological manifestation such as anxiety, confusion, irritability, hallucination, epilepsy, loss of consciousness and deep coma. If any patient is found to have neurological findings initially before involvement of other systems, had increased mortality of 20%[13], hypocalcemia can produce carpopedal spasm, paraesthesia, tetany or laryngeal stridor[25]

However, neural uptake of phosphorus is only small quantity [7], there was also no distinct histological change in the brain. Thus neurological manifestation was mainly due to hypocalcemia, rather than due to direct effect of phosphorus.

RENAL:

Half of the cases, have showed increase urea and creatinine values, but only few developed chronic kidney disease [27]. Rarely acute tubular necrosis also noted.

It mainly causes glomerular injury after absorption in GIT, histological finding shows fatty degeneration in few cases and fatty deposition with necrotic changes in some.

ELECTROLYTE DISTURBANCES:

Due to absorption of phosphorus and into conversion to phosphoric acid, some deproteination, hyperphosphatemia was noted. Calcium forms calcium phosphates salts which lead on to hypocalcemia. Hyperkalemia was noted, due to hypocalcemia or due to renal failure.

CHRONIC EXPOSURE:

It leads to mandibular osteonecrosis described as PHASSY JAW and also extreme fatigue and anaemia.

DIFFERENTIAL DIAGNOSIS :-

Unknown rodenticide exposure may be identified by few findings

Rapid action poisons, may manifests symptoms within six hours, if it shows more cholinergic findings, it suggests organophosphorus poisoning. More muscular activity suggests strychnine poisoning

More gastrointestinal symptoms such as mucosal burns, vomiting, abdominal pain and loose stools, suggests yellow

phosphorus poisoning. If it shows respiratory symptoms suggests zinc phosphide.

Delayed poisoning, manifests usually after twelve hours, most commonly due to superwarfarin groups [26]

The smell also shows some clue such as phosphorus smell as garlic; while zinc and aluminium phosphide smell as rotten fish, due to liberation of phosphine gas as it combines with air or water.

HEALTH CARE AWARENESS

Vomitus or stool containing white phosphorus is extremely dangerous, hence utmost care should be taken, to prevent toxicity to health care workers.

Few cases have also been reported, when these health care workers comes in contact with patient's stool or vomitus[27] .

Due to low melting point of phosphorus, it may present in clothes and other materials in liquid state and it may ignite at normal room temperature [28] .

Hence health care workers are advised to wear protective dress, in order to prevent them from contact with poison.

MANAGEMENT

INTIAL CARE:-

Basic life support such as protection of airway and circulatory status must be maintained.

Complete blood count , liver function test , prothrombin time , international standardised ratio (INR) , blood urea , serum creatinine , serum electrolytes especially of calcium phosphates , potassium should be done immediately after admission, On accessing intravenous line,

Frequent recording of vitals and cardiac monitoring should be done. Urine output should be recorded , if they complaint of irritation of eye , eye wash should be given with clean water immediately .

DECONTAMINATION OF SKIN:-

Patient with dermal exposure to poison should be immediately cleaned or immersed in water, it will reduce the wound size and hence reduce the hospital stay and also significantly reduce the death [29] .

Any region in skin which comes in contact with phosphorus, must be kept wet; to prevent ignition, if it gets contact with room oxygen [28] .

Copper sulphate solutions, which are usually used convert phosphorus element to less harmful – copper phosphate, which is black in colour and was easily removed from skin and clothes. But copper sulphate inhibits glucose – 6 – phosphate dehydrogenase enzyme, which may lead on to haemolysis, which turns on to be of worse prognosis [26] . It may also be identified by wood lamp examination, by fluorescent nature of phosphorus.

However by applying silver nitrate, it prevents phosphorus getting ignited by forming a protective silver layer over phosphorus [30] . After preventing ignition and identifying phosphorus, a through debridement of skin was done, since remaining poison may be prove to be toxic.

DECONTAMINATION OF DIGESTIVE TRACT:-

Early gastric lavage was indicated [31] . Due to high toxicity of phosphorus and since no specific antidote is available, lavage should be done in all patients. However utmost care must be taken, since explosion may occur following contact with oxygen, after inserting ryle's tube [8] . This may also be prevented by placing syringe filled with water at nasal end of the tube,after conforming the position of the tube , water is being instilled , instead of air .

However there are no datas available regarding supportive use of charcoal for adsorbing phosphorus , since due to it's high toxic nature and no oesophageal burns is getting reported , activated charcoal is being administered widely in many centres.

Bowel wash with polyethylene glycol is tried, since it forms non-absorbable material and thus getting washed away with stool [31]

Potassium permanganate wash will convert phosphorus to least toxic oxide [32] . Nowadays it is used worldwide, but anyway there was no positive results. It is not easily available and huge risk, it was not indicated.

TREATMENT:

No specific antidote is available up to date. But n-acetyl cysteine regimen have prevented death in significant number of patient, if it was administered early, if it was continued in full course. Since there is no potential side effect, no detailed study is available, if it is recommened to use in white phosphorus poisoning.

OTHER MODALITIES:

Steroids was not useful in preventing hepatotoxicity. In ubiquinone and sulphate were preventing liver toxicity to some extent, no human study is available yet [15].

HAEMODIALYSIS:

Haemodialysis rapidly correct the electrolyte disturbances such as hyperphosphatemia, hyperkalemia and hypocalcemia; but no report was available. However it significantly reduces mortality by 50%, thus exchange transfusion was beneficial.[10,14].

PHOSPHIDES:

Phosphides such as zinc and aluminium are being used as rodenticides in developing countries to protect grains from rodents [33], because it is cheaper and effective. Zinc phosphide is dark gray in colour, has rotten fish odour and bad taste, hence it was not used by other animals except rats. When mixed with tartar emetic, both these phosphides, release phosphine gas readily, when it comes in contact with water or diluted acids [33].

The exact mechanism is not clear. Phosphine is produced after phosphide reacts with hydrochloric acid in stomach. It inhibits

cytochrome-C oxidase and in turn inhibits oxidative phosphorylation of electron transport system [34], which in turn leads to various cellular toxicity, necrosis of gastrointestinal tract, liver and also kidneys. phosphine, being heavier than air, having rotten fish odour, can be detected even at levels of 2 ppm.[34] Typical odour can not be as warning sign, since toxicity occur even below the level of olfactory threshold. More number of oral exposures had been reported. It was also identified as one of the most common suicidal agent in India [35].

The exact level of lethal dose is not known since it was reported that even after ingesting a small amount of 5gm, the patient has died. Some patients have survived even after ingesting 50gm. Inhalation of toxic exposure of phosphine is also reported.

It causes severe mucosal irritation, manifested as nausea, vomiting and abdominal pain with in 15 minutes of ingestion of aluminium phosphide and 30minutes after ingestion zinc phosphide[35]

Other symptoms being hypotension, palpitation, acidosis, tetany. Systemic toxic symptoms such as broad QRS complex, jaundice and pulmonary edema is also reported. However neurological manifestations are rare, but seizures and coma are recorded in expired patients[36]. Majority of death occurs after 24 hours of ingestion, may be delayed upto 2 weeks, which was mainly reported due to myocardial damage [35].

Long term exposure in industry may reduce psychiatric, pulmonary, hepatic and cardiac findings.

MANAGEMENT:

Treatment is mainly supportive and also symptomatic. If there is acute ingestion, activated charcoal is useful, however patient should be continously monitor. Liver function test, urea, creatinine, electrolytes should be measured. After lavage, phosphine gas is emitted in lavage solution and stools, hence they must be cleaned and disposed properly to prevent inhalation exposure to health care workers by providing proper face masks.

Lavage are tried using sodium bicarbonate, plain water or even milk. Administration of proton pump inhibitor with antacid is useful. But prognosis is worse, if pulmonary and cardiovascular manifestations arise early. Chest x-ray for identifying pulmonary toxicity and abdominal x-ray for opacities in gut may help.

ANTI- COAGULANTS:

Warfarin and super warfarins belongs to this class of poison. Toxicity due to anticoagulants in both human and rodent are rare, it may require repeated exposure at small doses. No toxicity is reported in humans after single exposure. It was also least effective in rodents. Hence they

were considered as safe, but they are discarded as poor rodenticides. It leads to many resistant rats in few regions.

In 1980, however newer anticoagulants such as indanediones and hydroxy coumarins are produced. These are potent long acting, called “super warfarin”, produced significant anticoagulant effect for 6 weeks, even after single. Thus now it becomes one of deadliest toxin. It mostly occurs in children as accidental exposure [37]

MODE OF ACTION:

As warfarin is a known anti metabolite of vitamin k they inhibit synthesis of vitamin k dependent coagulation factors , namely ii ,vii , ix and x , by preventing the conversion of inactive vitamin K (epoxide)to its active form (quinol) . however repeated doses are required , which maintain maximum inhibition of active vitamin K synthesis , which results in elevated prothrombin levels , resulting in depletion of vitamin K dependent clotting factors and causes haemorrhage throughout the body , which may result in death . Bleeding occurs only if factors level, decreases more than one fourth of baseline level.

Since factor vii have short half life – 5 hours , rise in prothrombin time is seen in low half live – 20 hours , and will be rised till two days[37]

In addition, it also cause direct capillary damage, which leads to further bleed.

Initial test to be done are

1) prothrombin time,

2) International Normalised Ratio (INR)

3) complete blood count

4) liver function test

5) serum fibrinogen

6) measurement of vitamin K dependent coagulation factors

(ii , vii , viii , ix , x)

CLINICAL PRESENTATION:-

Patient is asymptomatic, immediately after ingesting poison, but develops symptoms 1 – 2 days after ingestion. They manifest various forms of bleeding manifestations in following orders of frequency as ecchymosis , haematuria , gynaecological bleeding , gastrointestinal bleeding , nasal bleed , haematoma , gum bleed .

However detailed investigations including prothrombin time, INR, blood count and even assessment of clotting factors such as ii and vii is useful. It is essential to measure this laboratory values, in helping to identify chronic ingestion of anticoagulants [38] .

Differential diagnosis being haemophilia, vitamin K deficiency, disseminated intravascular coagulation (DIC) and other factors deficiencies, other cause of liver failure must also be ruled out. In developed countries, measurement of newer agents level in blood as difenacoum is performed.

MANAGEMENT :-

It depends on time of presentation, amount ingested and bleeding manifestations. Acute intake of less than one bait of super warfarin does not produce significant toxicity and they are simply managed. Without gastric lavage and no lab investigations is needed; unless bleeding occurs.

If they ingest more than one bait, and present within four hours of ingestion, activated charcoal is administered.

Repeated administration of oral adsorbents such as cholestyramine , not only prevents absorption , but also reduces the half life of already absorbed anticoagulants.

Acute bleeding episodes are managed with crystalloids to replenish blood loss. Patients presenting with severe active bleeding are given fresh frozen plasma.

However, vitamin K (10mg) intravenously is given for adults. prophylactically, its action manifest only after 24 hours. It must be administered at less than 1mg/minute to reduce anaphylaxis. Repeated doses may be needed, which depends amounts and specificity of the compound. Prothrombin time should be monitor to evaluate if future treatment is needed.

A normal PT after two days excludes major anticoagulants activity [(39)].

All bleeding patients should be admitted to central bleeding, and patients with severe bleeding needs intensive care monitoring.

Asymptomatic patients are discharged immediately and follow up INR is carried out at 24 and 48 hours respectively.

All vitamin K preparations are not effective, phytonadione(vitamin K1) is used for this purpose. The needed dose, duration of treatment varies widely with amount and type of poison taken and also patient metabolism.

Taking serial measurements of toxin level, determines duration of treatment needed.

Maximum dose of 100mg in a day for upto 10 months is being used in huge bleeding [39].

Now, let us see about the other compounds

HIGHLY TOXIC COMPOUNDS:

Signal word: DANGER, (LD 50 <50mg/kg)

THALLIUM:-

Physical nature: white colour, crystalline in nature, odourless and Tasteless.

Mechanism [41] : it combines with mitochondrial sulphydryl groups and Thus interfering with the oxidative phosphorylation of Respiratory chain

Fatal dose : 14 mg/kg.

Volume of distribution : 3.6 L/kg in humans ; 20 L/kg in rats

Half life : 2 to 15 days [40]

Clinical symptoms & signs: anorexia, abdominal pain, loose stools , Delirium, coma, seizures, later symptom

Of alopecia.

Onset of symptoms: gastrointestinal symptoms develop immediately after Ingestion, but other symptoms may develop after 12 hours.

Antidote : activated charcoal and ferrocyanides .

FLOUROACETATE:-

Physical nature: White colour, crystalline in nature, odourless and Tasteless, water soluble

Mechanism : i) flouroacetate gets converted to flourocitrate , which Interfers with kreb's cycle [42]

ii) sodium flouroacetamide

Fatal dose : i) flouroacetate :- 3- 7 mg / kg

ii) sodium flouroacetamide :- 13-14 mg /kg

Clinical signs& symptoms: seizures, tachycardia – mainly ventricular and coma

Onset of symptoms : 2 to 20 hours

Prognostic indicators : hypotension, increased creatinine level and acidosis, may lead to death , hypotension is due to decreased systemic vascular resistance and increased cardiac output .

Antidote : i) no specific antidote

ii) activated charcoal and sorbitol are recommended .

iii) 500 ml of 10% acetamide in 5% dextrose over 30 mins every 4 hours or 10% ethyl alcohol solution are used with limited Success [43] .

STRYCHININE:-

Physical nature : bitter in taste

Mechanism of toxicity: competitive antagonist for glycine at post synaptic

Spinal motor neuron [44] .

Fatal dose : for children – 15 mg [45] .

for adults - 1-2 mg/kg .

Volume of distribution: 13L/ kg

Clinical signs and symptoms : anxiety, restlessness, hyperextended

posture, locked jaw, risus sardonicus , dysphagia.

Onset of action : 10 to 20 minutes.

Antidote and treatment: quiet room,

Intravenous benzodiazepines and

Neuromuscular blockade drugs.

ARSENIC TRIOXIDE

Physical nature : White colour , crystalline in nature .

Mechanism of toxicity : it combines with sulphydryl groups and thus Interfers with various enzymatic actions.

Fatal dose : 1- 4 mg/kg

Clinical signs and symptom: difficulty in swallowing , nausea , vomiting , Bloody diarrohea , cardiac collapse and Altered sensorium .

Onset of action : for symptoms - 1 hour.

For death : maximum of 24 hours.

Antidote : i) succimer and dimercaprol [46] until urine arsenic Level is less than 50 microgram in 24 hours.

ii) haemodialysis.

BARIUM

(available in soluble form as carbonate , chloride)

Physical nature : yellow colour, lustrous lump.

Mechanism of toxicity: hypokalemia and

Neuromuscular blockade.

Fatal dose : 20-30 mg/kg.

Clinical symptoms & signs: headache, paraesthesia , paralysis , nausea ,

Vomiting, loose stools, ECG changes as

Arrhythmias and cardiac failure

Onset of action : 1-8 hours

Antidote : i) orogastric lavage with sodium sulphate [47]

ii) potassium supplementation .

N-3- Pyridyl methyl –N`-p-Nitro-phenyl Urea (PNU)

Physical nature : yellow colour, like yellow-green powder in bait.

Mechanism of toxicity : Interfers with nicotinamide metabolism in pancreas and destroys beta cells [48] , nervous system and heart.

Fatal dose : 5 mg/kg.

Clinical signs & symptoms: vomiting with abdominal pain, severe Orthostatic hypotension, hyperglycaemia [49] , Neuropathy and pneumonia.

Onset of action : maximum of 2 days.

Antidote : i) Nicotinamide 500 mg IV – loading dose and Then titrate, according to toxicity.

ii) Manage hyperglycaemia with insulin .

MODERATELY TOXIC COMPOUNDS

Signal word: WARNING (LD 50 - 50 to 500 mg/kg)

ALPHA – NAPHTHYL THIO UREA (ANTU)

Physical nature : odourless, slightly bitter in taste, fine – gray powder, insoluble in water.

Mechanism of toxicity: acute lung injury[50] .

Clinical symptoms and signs: breathing difficulty, rales , froth , cyanosis , hypothermia.

Onset of action : not estimated.

Antidote : only supportive care.

CHOLECALCIFEROL (VITAMIN D3)

Physical nature : as various sized pellets.

Mechanism of toxicity: hyper calcemia.

Fatal dose : not estimated

Clinical symptoms and signs: headache, lethargy, weakness, renal failure, metastatic calcification.

Onset of action : hours to days.

Antidote : i) fluid balance.

ii) frusemide, prednisolone , calcitonin , biphosphates .

LOW TOXICITY COMPOUNDS

Signal word :- CAUTION (LD 50 , 500 to 5000 mg/kg)

RED SQUILL

Physical nature : bitter taste

Mechanism of toxicity: as cardiac glycoside [51] .

Onset of action : 30 minutes to 6 hours.

Fatal dose : not estimated.

Clinical symptoms & signs: myocardial irritability, blurred vision, Hyperkalemia.

Antidote : digoxin specific antibodies, atropine.

NORBROMIDE

Physical nature : yellow cornmeal bait;

peanut butter.

Mechanism of toxicity: vasoconstriction and ischaemia in rats through specific

smooth muscle receptors.

Clinical symptoms and signs: transient hypothermia and hypotension[52)] .

Fatal dose and onset of action: not estimated.

Antidote : only supportive care.

BROMETHALIN

Physical nature : 7.5% concentrate pellets.

Mechanism of toxicity : uncouples oxidative phosphorylation [53] ,

Interrupts nerve conduction.

Clinical symptoms and signs: muscle tremor, myoclonic jerks,

ataxia, coma, focal seizures.

Immediate onset of action .

Only supportive care .

SHORT ACTING ANTICOAGULANTS

WARFARIN & PROLIN:

Physical nature : yellow cornmeal or rolled oats.

Mechanism of toxicity: warfarin – anticoagulation by vitamin k dependent Factors (ii,vii, ix ,x); death due to haemorrhage.

Prolin - combines with antibiotic and eliminates intestinal organisms that produce vitamin k.

Signs : bleeding with elevated INR values.

Onset of action : 12 to 48 hours.

Antidote : vitamin k1 and fresh frozen plasma (FFP).

OTHER LONG ACTING ANTICOAGULANTS

INDANDIONES:

Pindone, Pivalyn, Valone, Diphacinone.

Physical nature : mouldy, fluffy yellow powder.

Mechanism of toxicity: anticoagulant.

Onset of action : several days.

Clinical symptoms & signs: chronic ingestion produces cardiac and Cardiac and neurological symptoms, along bleeding with elevated INR.

Antidote : vitamin k1 and FFP.

MATERIALS AND METHODS:-

This study was conducted at Thanjavur medical college, Thanjavur , during the period of January 2012 to November 2012 . There were 108 patients admitted with alleged history of ingesting rodenticide compound in our emergency medical ward during the study period. After applying, the inclusion and exclusion criterias , 60 patients only fulfilled all the criteria and they were chosen as study subjects (n =60).

Inclusion criteria:-

1. Patient ingesting rodenticide (bait, paste or powder) immediately before admission, maximum period of within 24 hours.

Exclusion criteria:-

1. Patient treated outside and referred to our hospital.
2. Patient presenting after 24 hours of ingestion.
3. Patient consuming the poison along with alcohol or other compounds.
4. History of chronic alcoholism or jaundice in the recent past.
5. Suffering from any other serious systemic diseases.

Method of collection of datas:-

Patient admitted in emergency medical ward with history of ingesting rodenticide compound were taken as study subjects. A complete history, clinical examination of toxicity of individual compounds and certain relevant biochemical investigations were performed .patients fitting into the inclusion criteria as described by the patients or identification of rodenticide compound.

After obtaining complete history, a complete clinical examination was performed, with particular importance to the vitals, jaundice, bleeding manifestations, as explained in the proforma. This evaluation was done in our emergency ward. Patient was further followed till their stay in hospital, they are noticed for development of jaundice or further prognosis of the patient and relevant investigation datas are collected.

Importance to jaundice , bleeding tendencies , oliguria and seizures or headache , serum bilirubin value on admission , on 4 th day and afterwards , INR value , serum creatinine and SGOT value on 4 th day were collected .

All patients were managed with usual decontamination method including nasogastric lavage and treated according to nature of compound

with intramuscular vitamin k and laxatives. inj N-acetyl cystiene was tried in intensive care unit .

Counselling was given to the survivors.

Autopsy was done for all expired patients.

Statistical tests:-

Chi square test and one way ANOVA ‘f’ test were used in this study.

Ethical committee clearance was obtained before commencing the study.

Informed consent is obtained either from patients or their relatives.

RESULTS

FIGURE 1 : AGE DISTRIBUTION

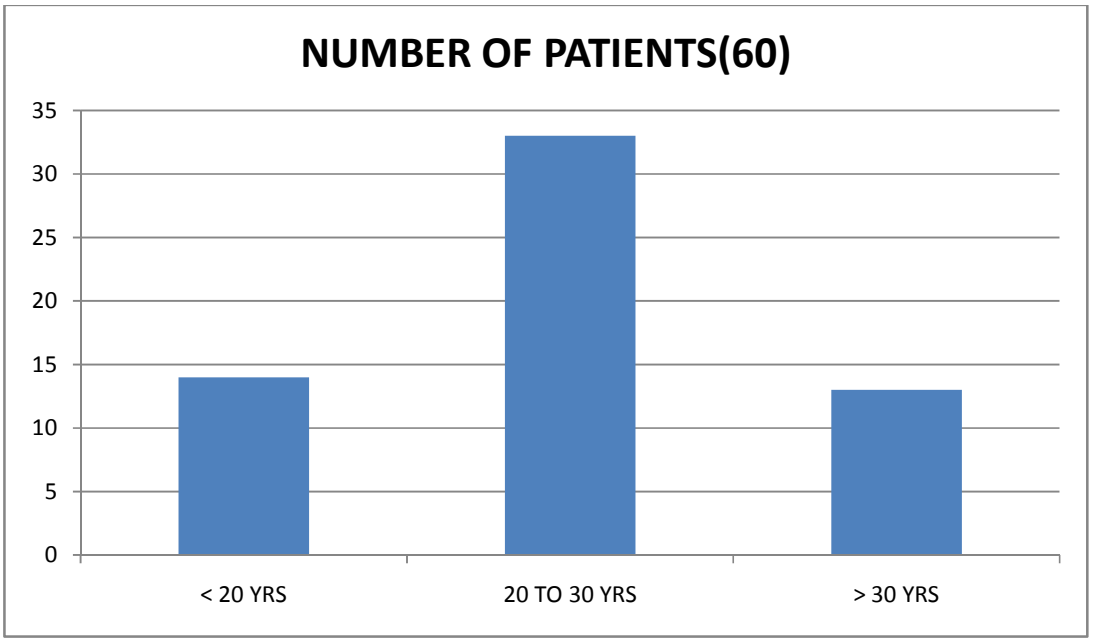
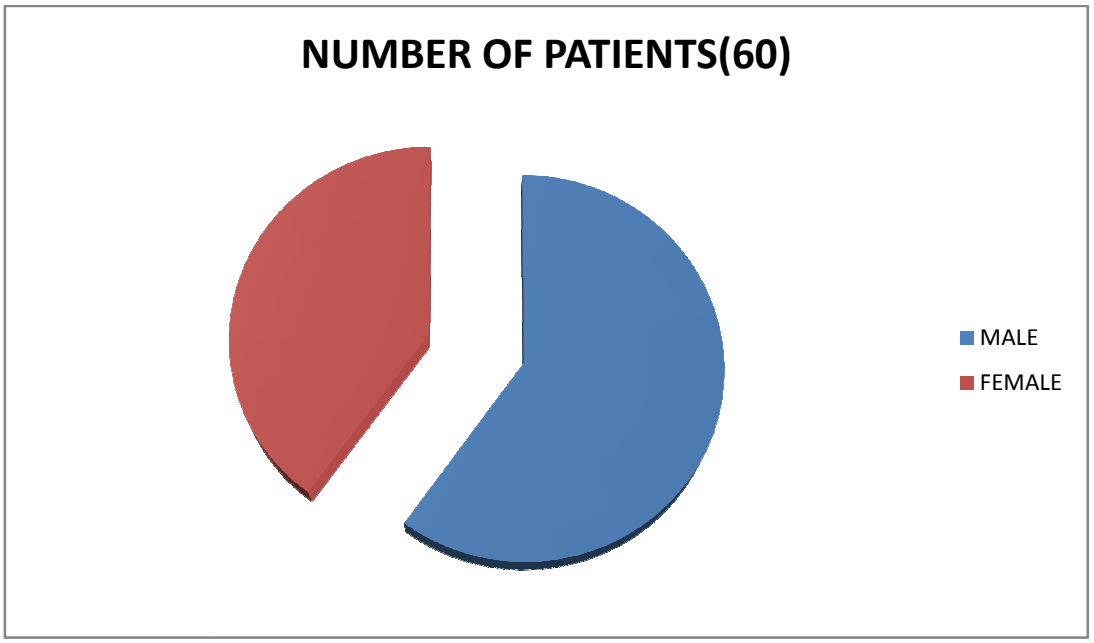


TABLE 1 :

AGE (YRS)	NO OF PATIENTS(60)
LESS THAN 20	14
20 TO 30	33
MORE THAN 30	13

Patients are most common in 20 – 30 years of age group.

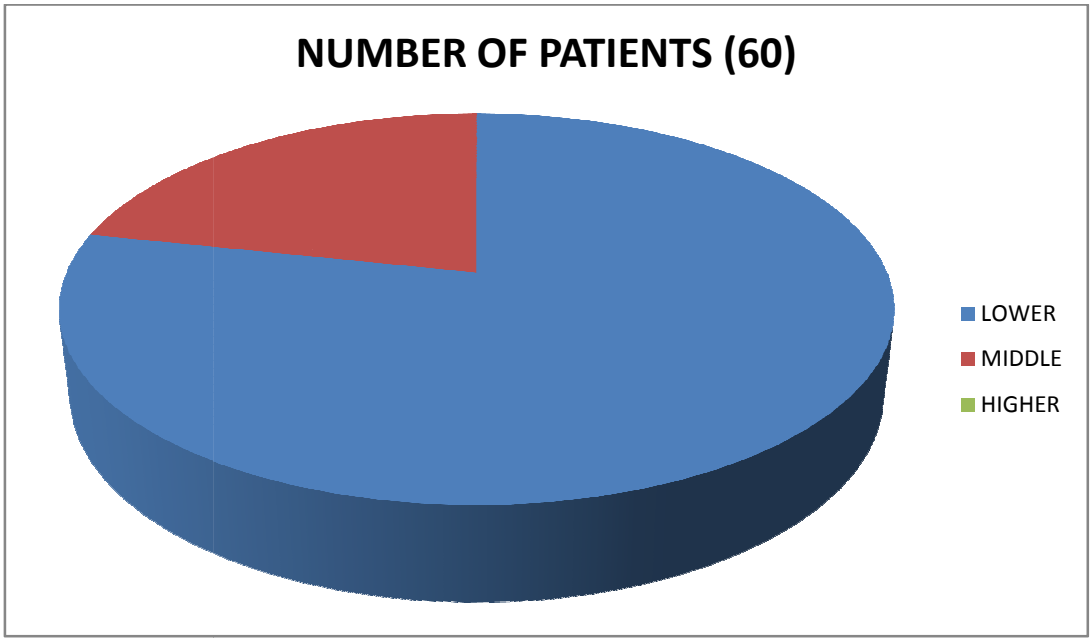
FIGURE 2: SEX DISTRIBUTION



MALE - 36 PATIENTS (60%)

FEMALE - 24 PATIENTS (40%)

FIGURE 3: ACCORDING TO SOCIO-ECONOMIC STATUS :-

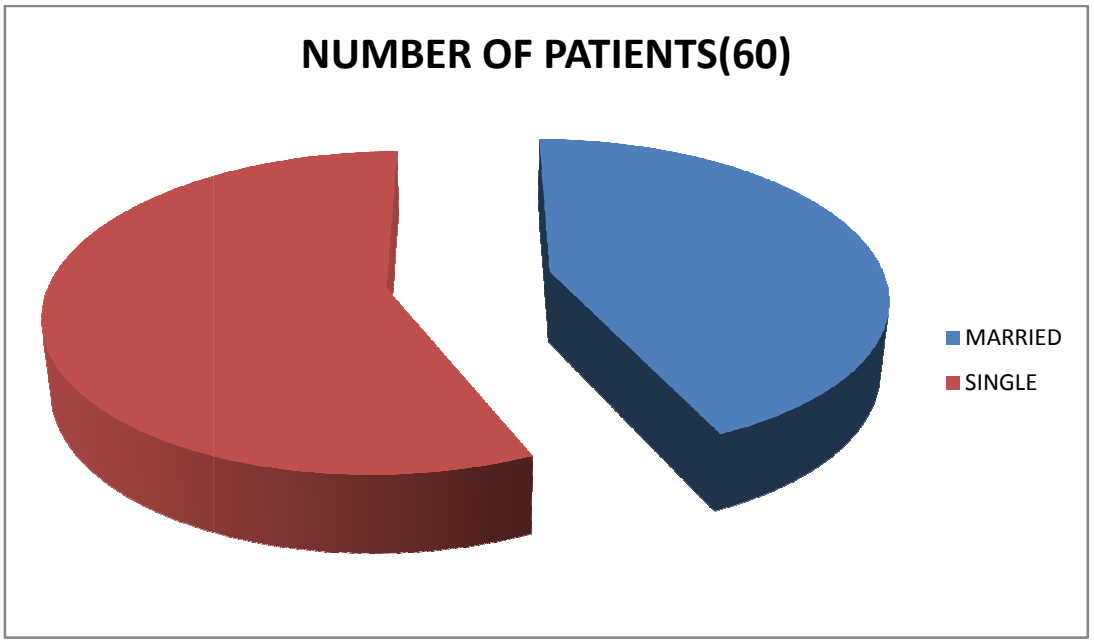


LOWER SOCIO-ECONOMIC STATUS :- 47 Patients (78.3%)

MIDDLE SOCIO-ECONOMIC STATUS :- 13 Patients (21.7%)

HIGHER SOCIOECONOMIC STATUS :- 0 Patients (0%)

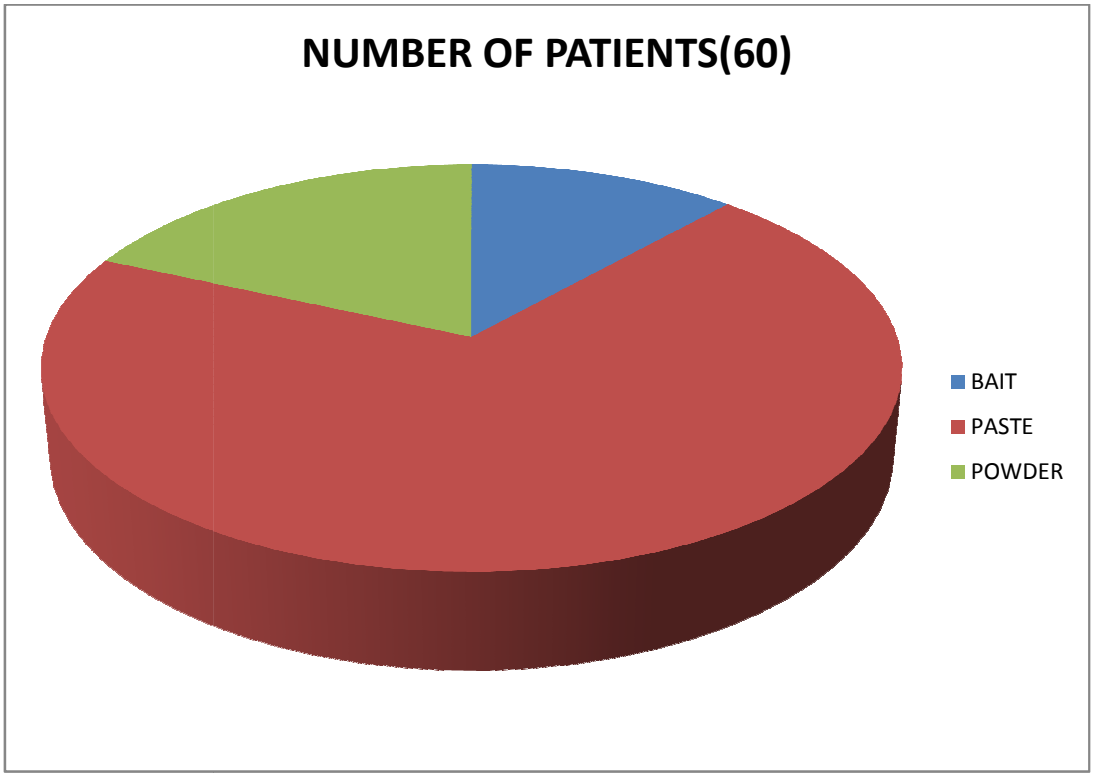
FIGURE 4 : MARITAL STATUS OF THE PATIENT :-



MARRIED PERSONS :- 26 (43.3%)

UNMARRIED PERSONS :- 34 (56.7%)

FIGURE 5 : QUALITY OF INGESTED POISON :-

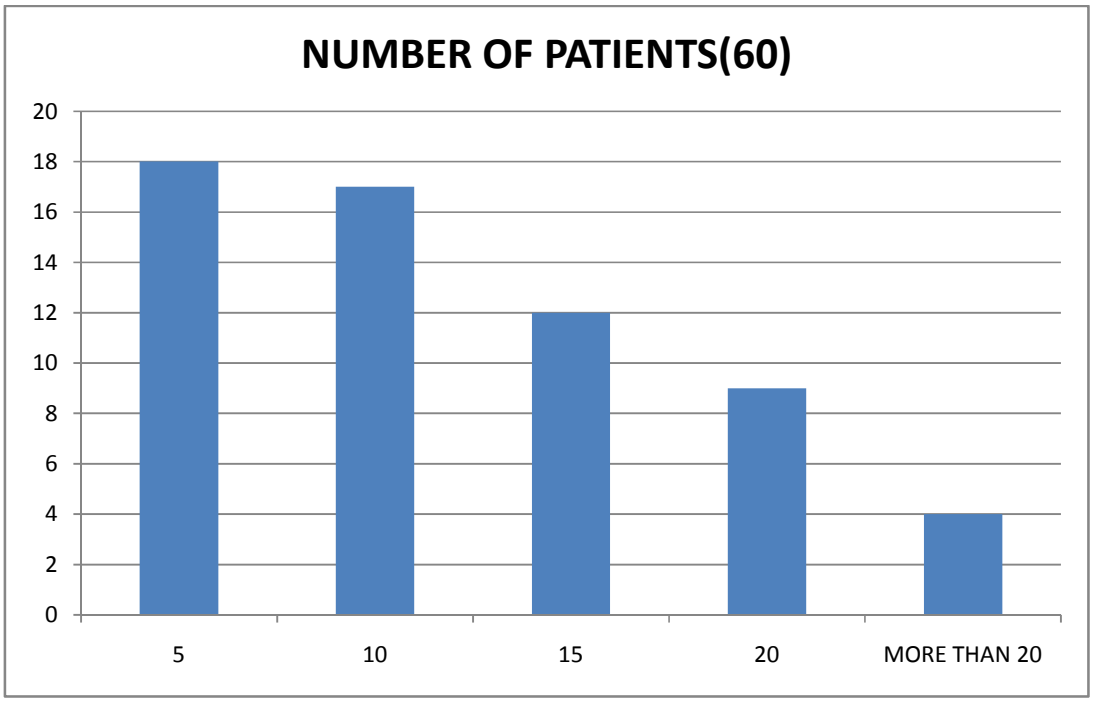


BAIT :- 7 Patients (11.7%)

PASTE :- 42 Patients (70.0%)

POWDER :- 11 Patients (18.3%)

FIGURE 6: QUANTITY OF POISON CONSUMED :-



5 gms :- 18 patients (30.0%)

10 gms :- 17 patients (28.3%)

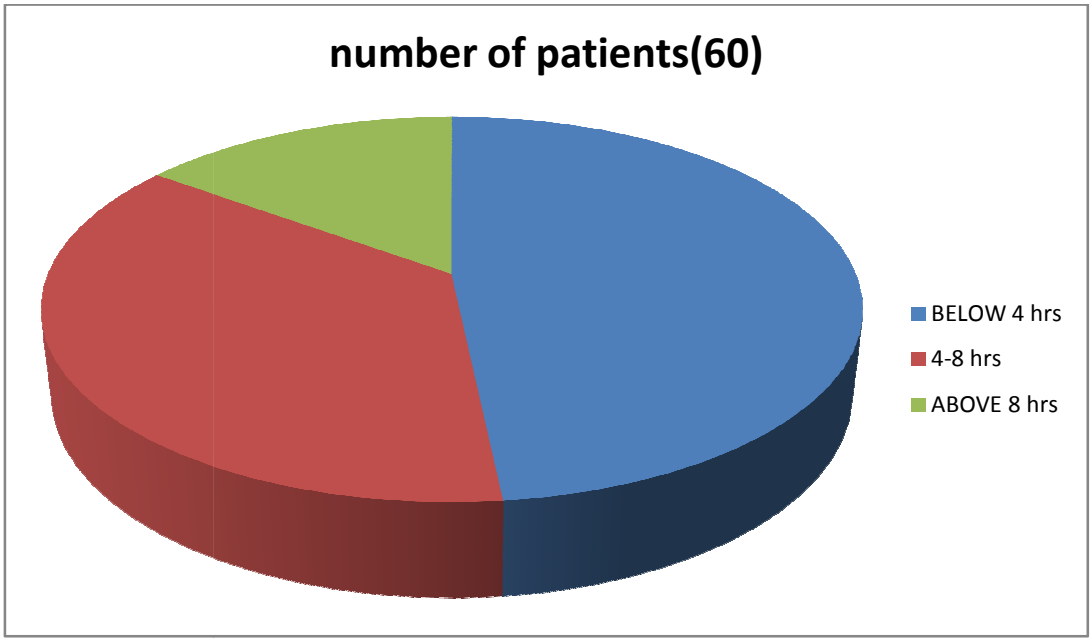
15 gms :- 12 patients (20.0%)

20gms :- 9 patients (15.0%)

25gms :- 3 patients (5.0%)

30gms :-1 patients (1.7%)

FIGURE 7 : TIME DELAY OF PATIENT FROM INGESTION TO HOSPITALISATION :



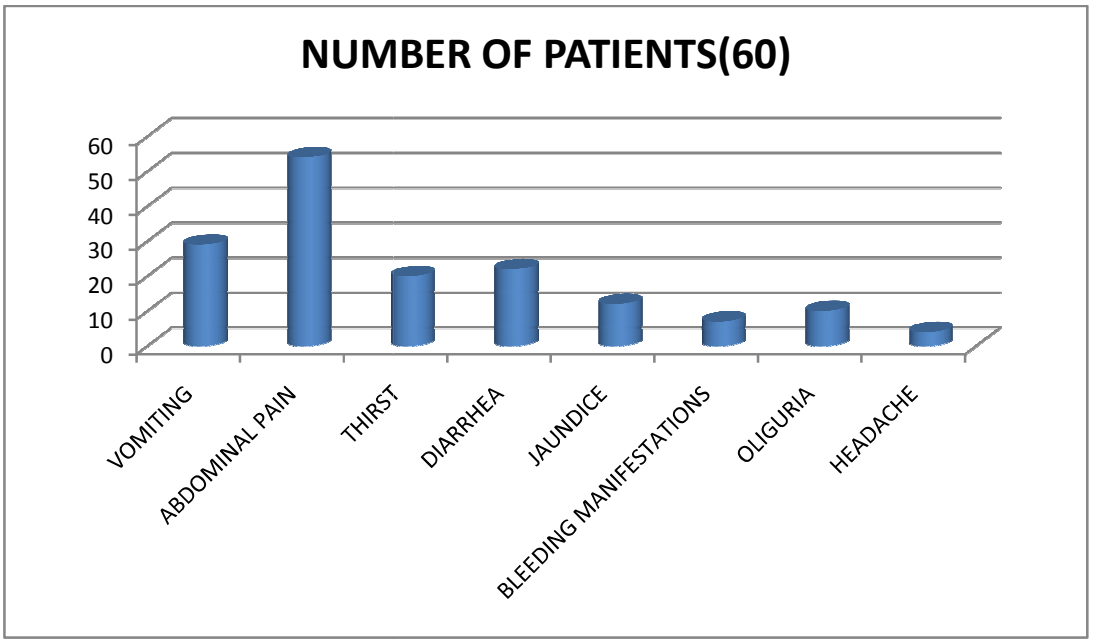
BELOW 4 HRS :- 29 (48.3%)

4 TO 8 HRS :- 22 (36.7%)

ABOVE 8 HRS :- 9 (15%)

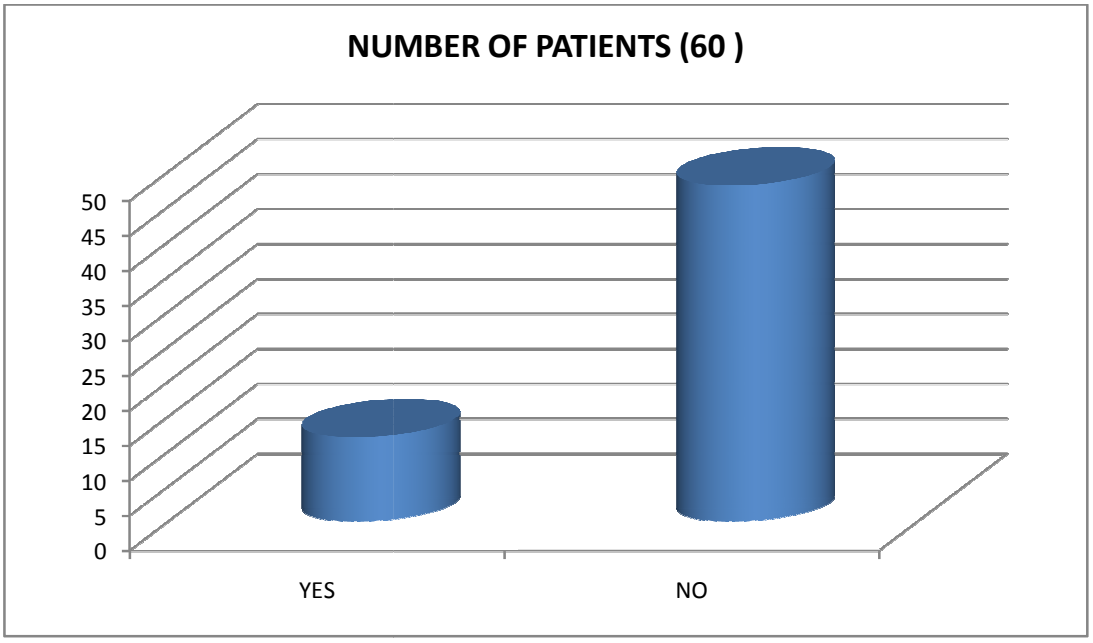
CLINICAL MANIFESTATIONS :

FIGURE 8 : Comparison of symptomatology



Thus commonest symptom is abdominal pain , observed in 54 patients (90%) , followed by vomiting which is found in 29 patients (48.3%) , next being diarrhoea which is observed in 22 patients (36.7%) . however jaundice is observed in 12 patients , still mortality is observed in all patients of jaundice (100%) .

FIGURE 9 : JAUNDICE DEVELOPED AFTER ADMISSION



Patient developing jaundice after admission , mostly on 3rd or 4th day . almost all

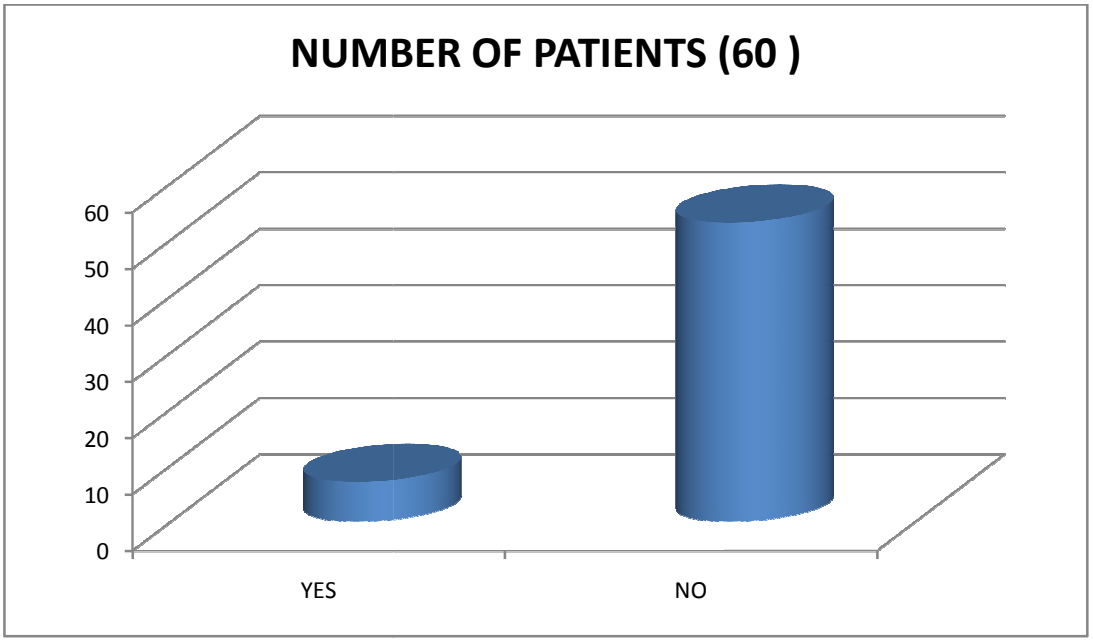
Of the patients , were ingested paste (phosphorus) and every patient was expired soon . this explains the main cause of death and also

The most dangerous compound .

YES : 12 (20.0%)

NO : 48 (80.0%)

FIGURE 10 : BLEEDING MANIFESTATIONS :



Patients developing bleeding manifestations after ingesting poison ,

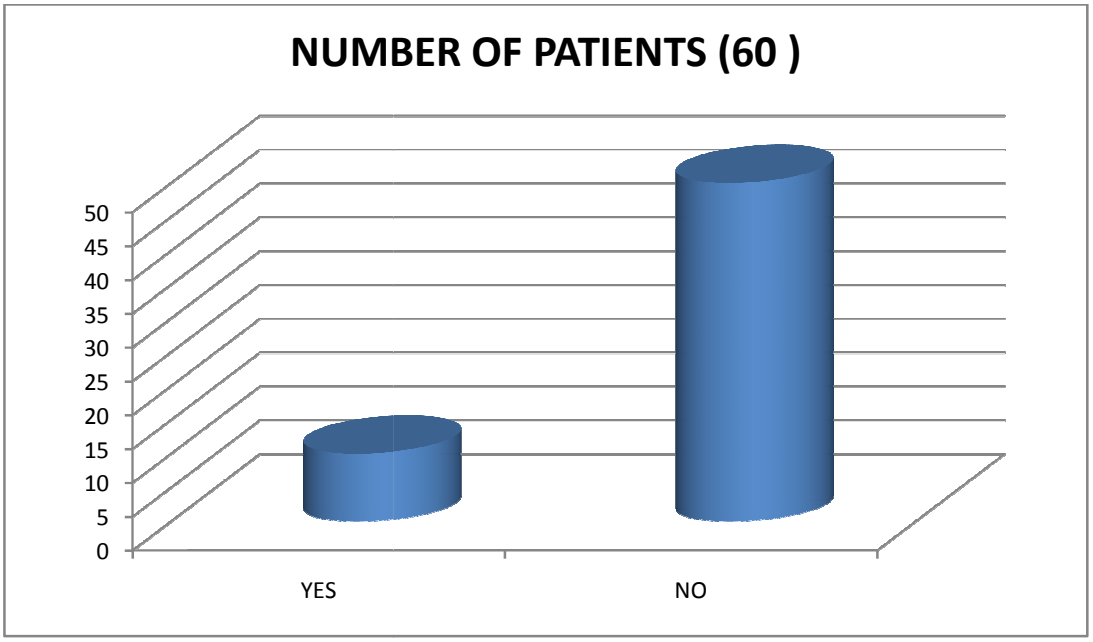
Of 7 pts with bleeding , 5 had consumed bait (hydroxyl coumarins) and other two consumed paste (phosphorus) probably due to acute hepatic toxicity .

patients in bait group , had recovered after inj vit k for 3 days.

Yes : 7 (11.7%)

No : 53 (88.3%)

FIGURE 11 : OLIGURIA



Of 10 patients develop oliguria after admission , all of them belongs

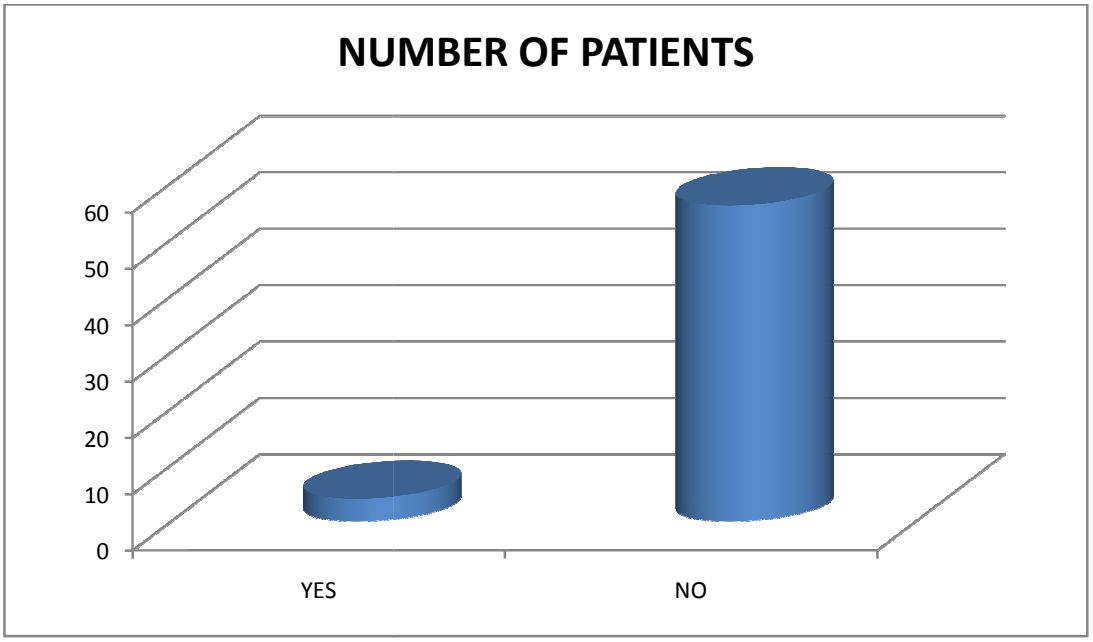
To paste (phosphorus) group , and all of them were expired . this explains

That toxicity was also due to renal injury

YES : 10 (16.7%)

NO : 50 (83.3%)

FIGURE 12 : HEADACHE

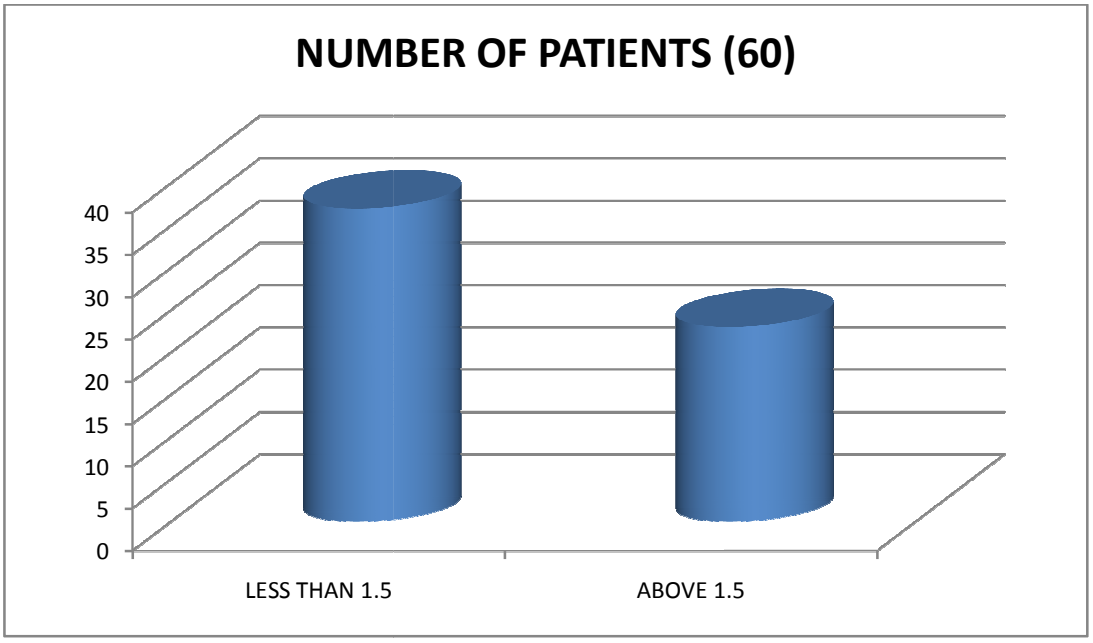


Only 4 patients had developed headache , no other neurological complications , all 4 belongs to paste (phosphorus) group , and all of 4 patients had expired .

YES : 4 (6.7%)

NO : 56 (93.3 %)

FIGURE 13 : SERUM BILIRUBIN LEVEL ON 4TH DAY

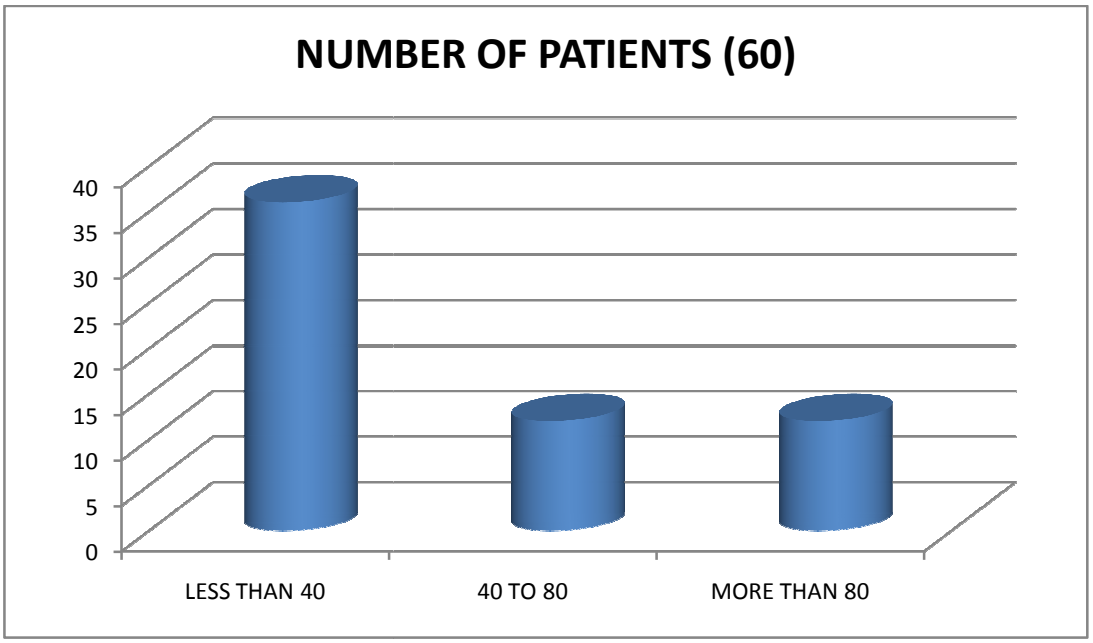


By estimating serum bilirubin level on 4th day of admission , 23 patients showed bilirubin more than 1.5 mg , almost all of them had ingested Paste (phosphorus) . out of which 12 patients had expired .

LESS THAN 1.5 mg : 37 (61.7%)

ABOVE 1.5 mg : 23 (38.3%)

FIGURE 14 : SGPT LEVEL ON 4TH DAY OF ADMISSION



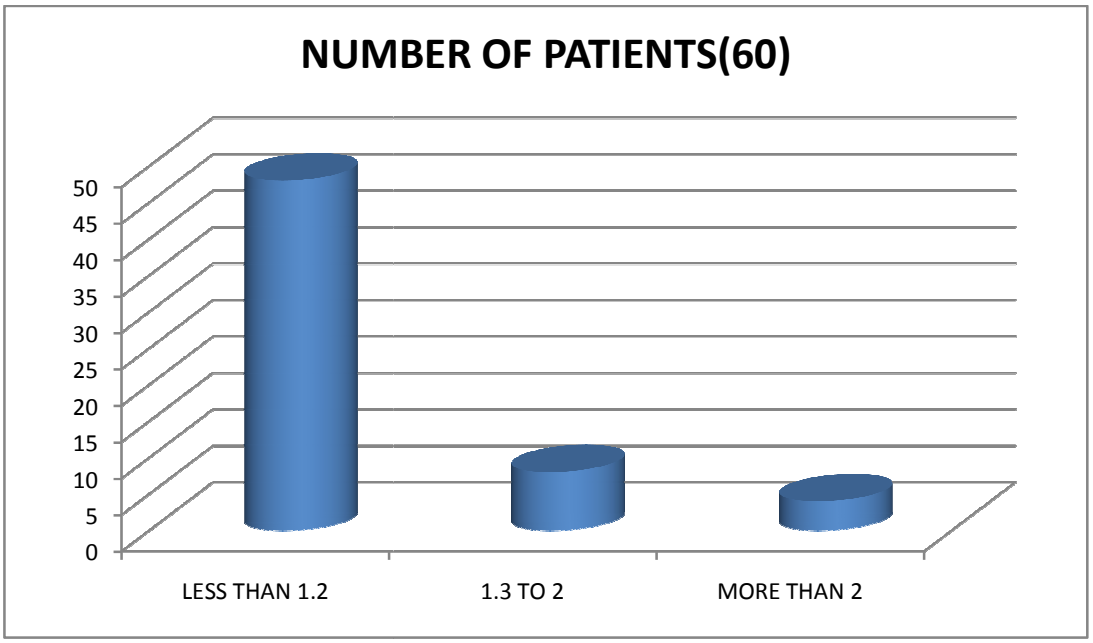
Of total 60 patients , 24 patients showed SGPT values of more than Normal value (40iu/l) , which almost identical with elevated bilurubin level . 12 patients had values more than five to ten times normal value , indicates acute Hepatocellular failure , it carries almost 100% mortality .

LESS THAN 40IU/L : 36 (60%)

40 TO 80 IU/L : 11 (18.5%)

MORE THAN 80IU/L : 13 (21.5%)

FIGURE 15 : SERUM CREATININE VALUE OF THE PATIENTS



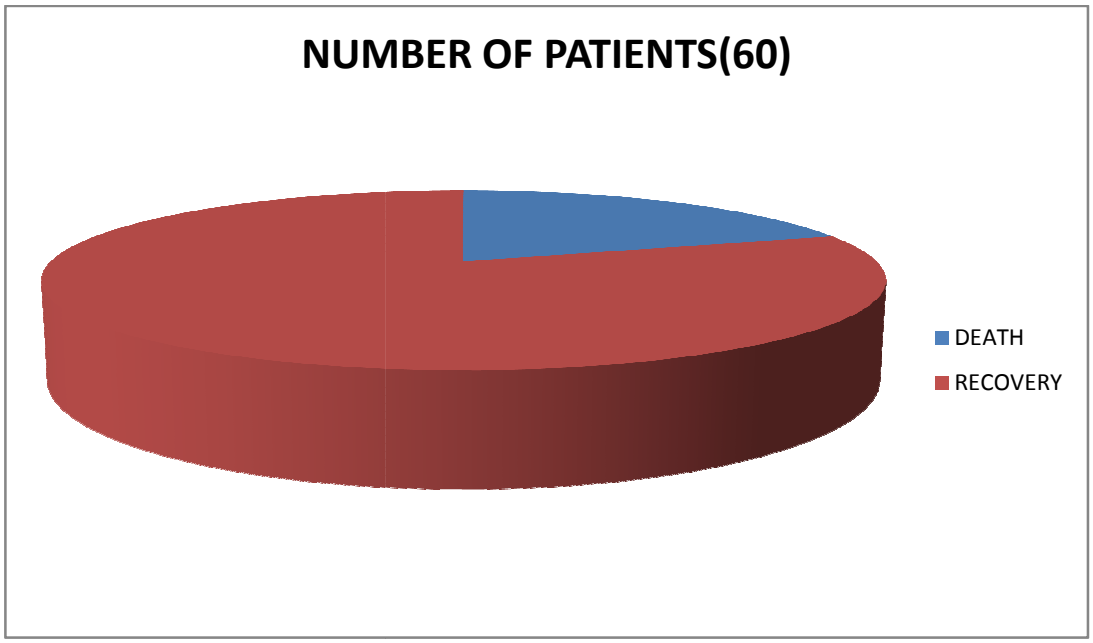
Of total patients , 12 patients shows elevated serum creatinine Values , which indicates the renal toxicity of the phosphorus poison , which Also carries the worse prognosis .

LESS THAN 1.2 mg /dl : 48 (80%)

1.3 TO 2 mg/dl : 8 (13.3%)

MORE THAN 2 mg/dl : 4 (6.7 %)

FIGURE 16 : RECOVERY OF THE PATIENT



Of 60 patients got admitted , 12 patients had expired, all of Them had consumed paste (phosphorus) , thus indicating the lethal mode of poison .

DEATH : 12 (20 %)

RECOVERY : 48 (80 %)

FIGURE 17 : AGE DISTRIBUTION COMPARED WITH MORTALITY :

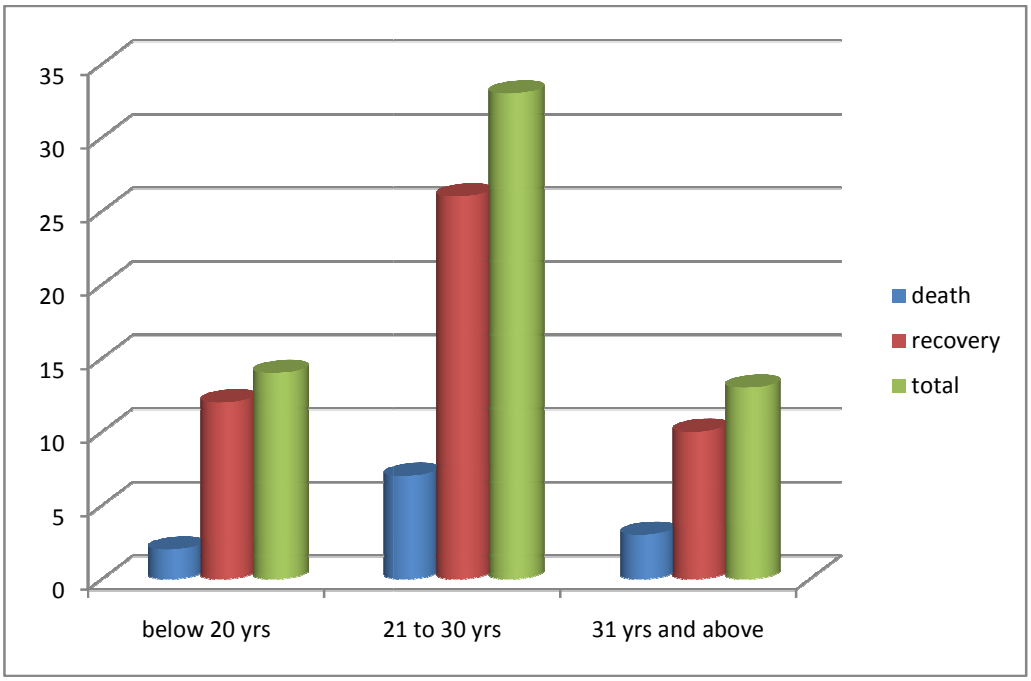


TABLE 2 :

AGE GROUP (YEARS)	DEATH (100%)	RECOVERY(100%)	TOTAL(100%)
BELOW 20	2 (16.7%)	12 (25%)	14 (23.3%)
21 TO 30	7 (58.3%)	26 (54.2%)	33 (55%)
ABOVE 31	3 (25%)	10 (20.8%)	13 (21.7%)

FIGURE 18 : SEX DISTRIBUTION WITH MORTALITY :

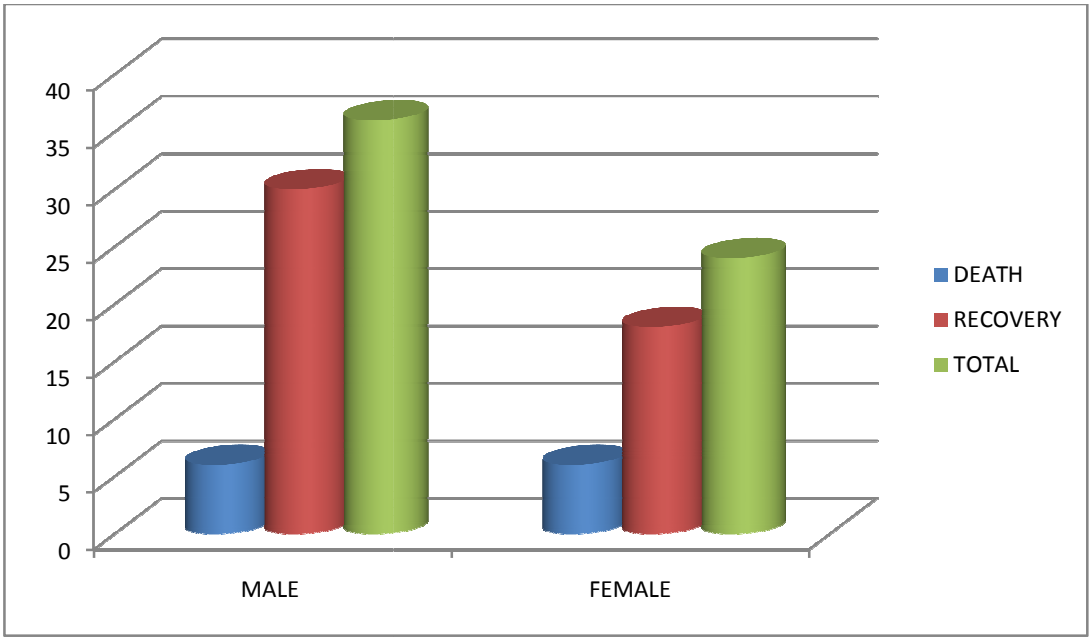


TABLE 3 :

SEX	DEATH	RECOVERY	TOTAL
MALE	6 (50%)	30 (62.5%)	36 (60%)
FEMALE	6 (50%)	18 (37.5%)	24 (40%)

THUS FEMALE AND MALE PATIENTS HAVE EQUAL MORTALITY , HOWEVER MALE GOT MORE ADMISSION THAN FEMALE

FIGURE 19 : SOCIO-ECONOMIC STATUS WITH MORTALITY

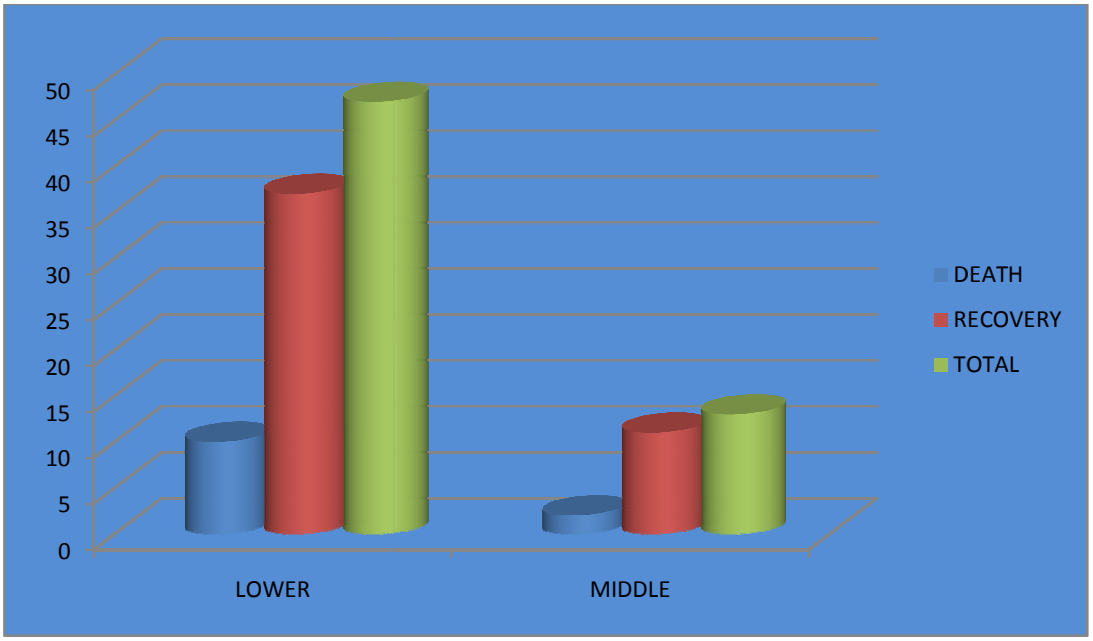


TABLE 4 :

SOCIO-ECONOMIC	DEATH	RECOVERY	DEATH
LOWER	10 (83.3%)	37 (77.1%)	47 (78.3%)
MIDDLE	2 (16.7%)	11 (22.9%)	13 (21.7%)

BOTH INCIDENCE AND MORTALITY WAS

COMMONER IN PATIENTS FROM LOW COCIO-ECONOMIC STATUS.

FIGURE 20 : MARITAL STATUS COMPARED WITH MORTALITY:

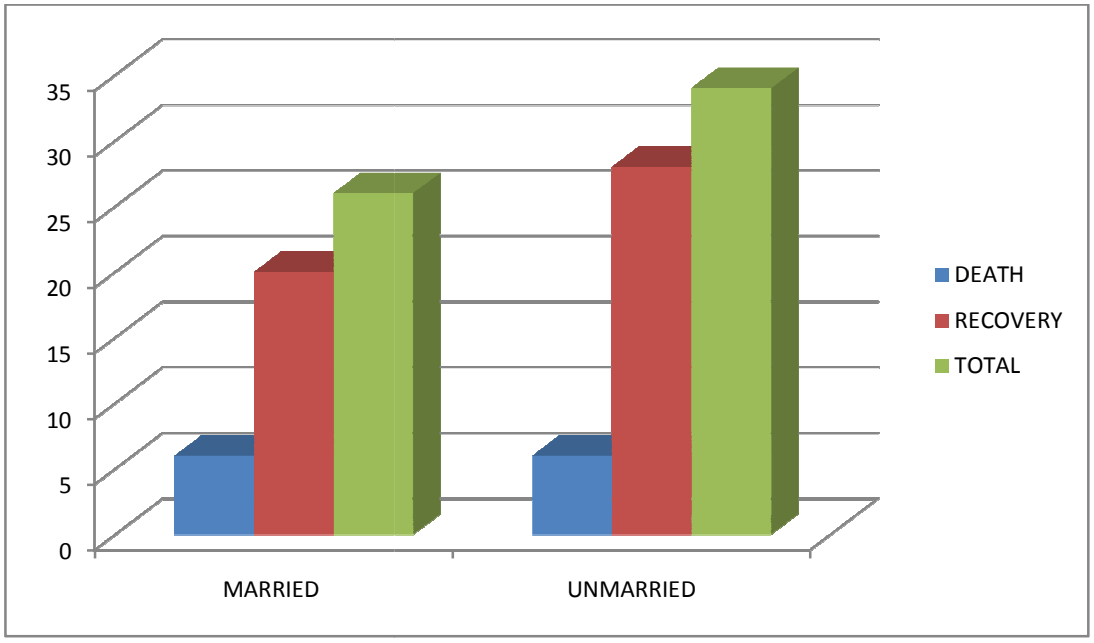


TABLE 5 :

MARITAL STATUS	DEATH	RECOVERY	TOTAL
MARRIED	6 (50%)	20 (41.7%)	26 (43.3%)
UNMARRIED	6 (50%)	28 (58.3%)	34 (56.7%)

FIGURE 21 : QUALITY OF THE POISON WITH MORTALITY :

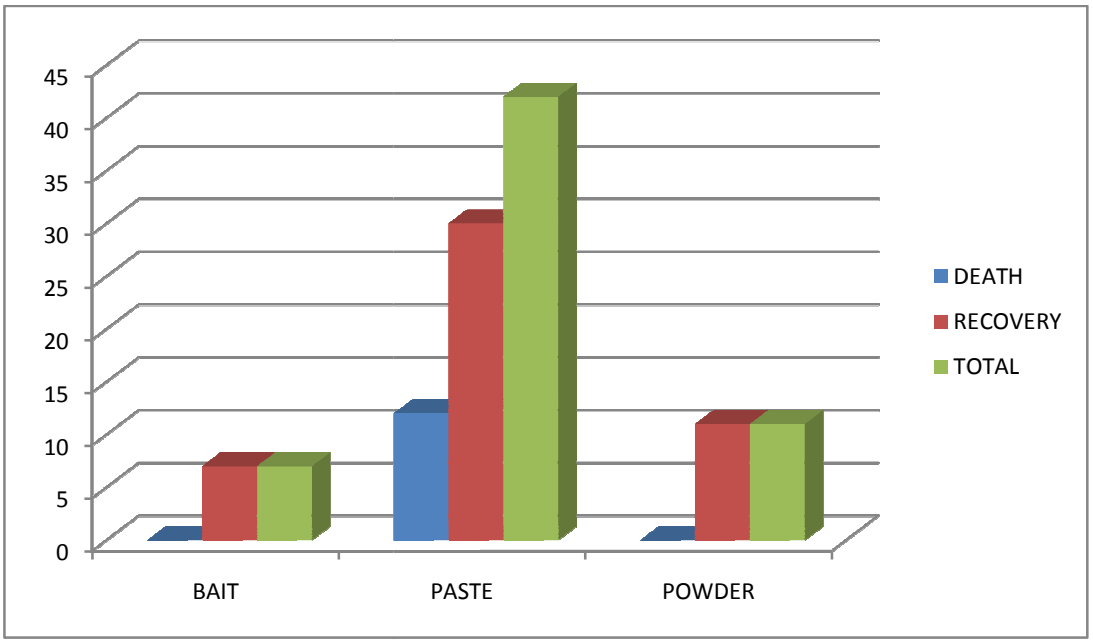


TABLE 6:

QUALITY	DEATH	RECOVERY	TOTAL
BAIT	0 (0%)	7 (14.6%)	7 (11.7%)
PASTE	12 (100%)	30 (62.5%)	42 (70 %)
POWDER	0 (0%)	11 (22.9%)	11 (18.3%)

Mortality is only with phosphorus compound (paste).

FIGURE 22 : TIME DELAY WITH MORTALITY :

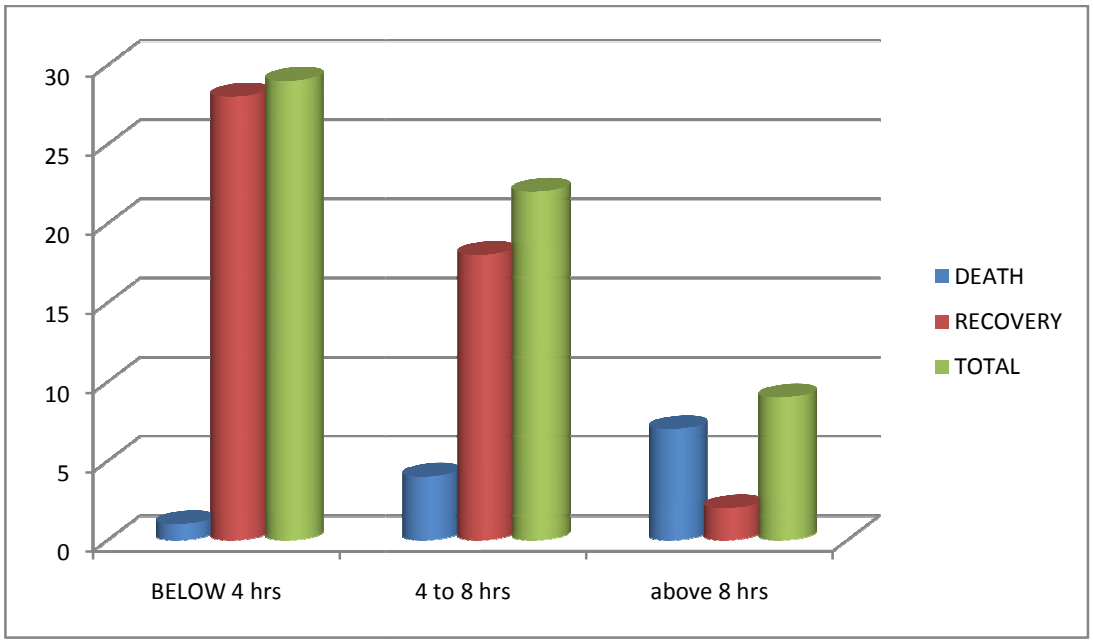


TABLE 7 :

TIME DELAY (hrs)	DEATH	RECOVERY	TOTAL
BELOW 4	1 (8.3%)	28 (58.3%)	29 (48.3%)
4 TO 8	4 (33.3%)	18 (37.5%)	22 (36.7%)
ABOVE 8	7 (58.3%)	2 (4.2%)	9 (15.0%)

More time delay causes comparitevely more mortality .

FIGURE 23 : JAUNDICE COMPARED WITH MORTALITY :

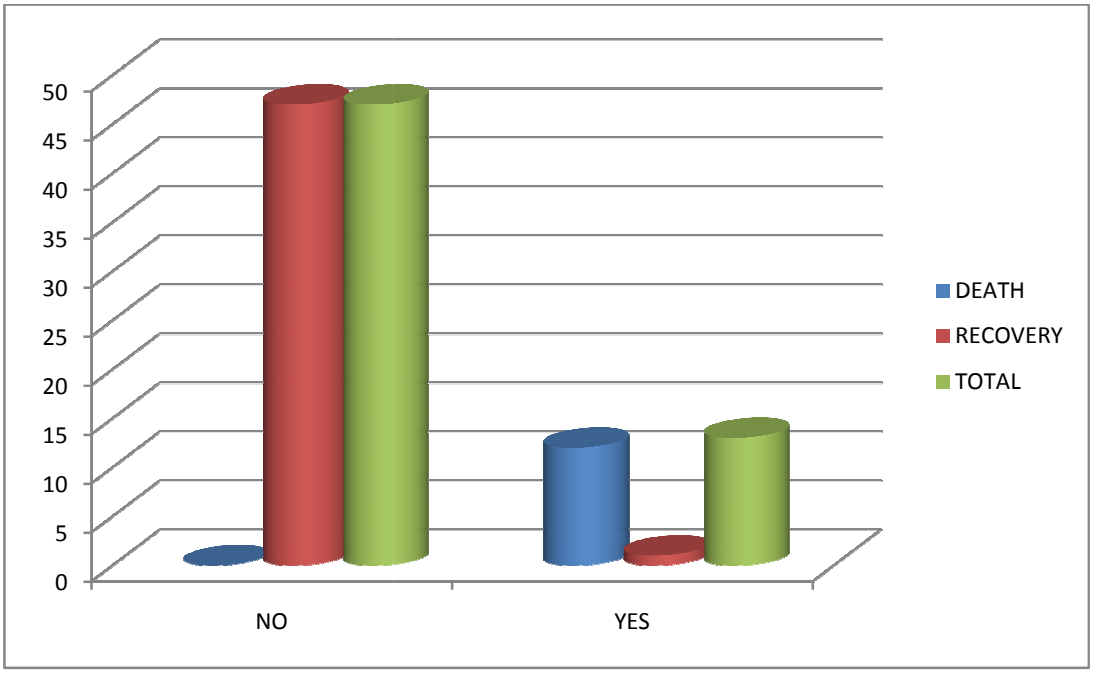


TABLE 8 :

JAUNDICE	DEATH	RECOVERY	TOTAL
NO	0 (0 %)	47 (97.9 %)	47 (78.4%)
YES	12 (100%)	1 (2.1%)	13 (21.6%)

FIGURE 24 : OLIGURIA WITH MORTALITY :

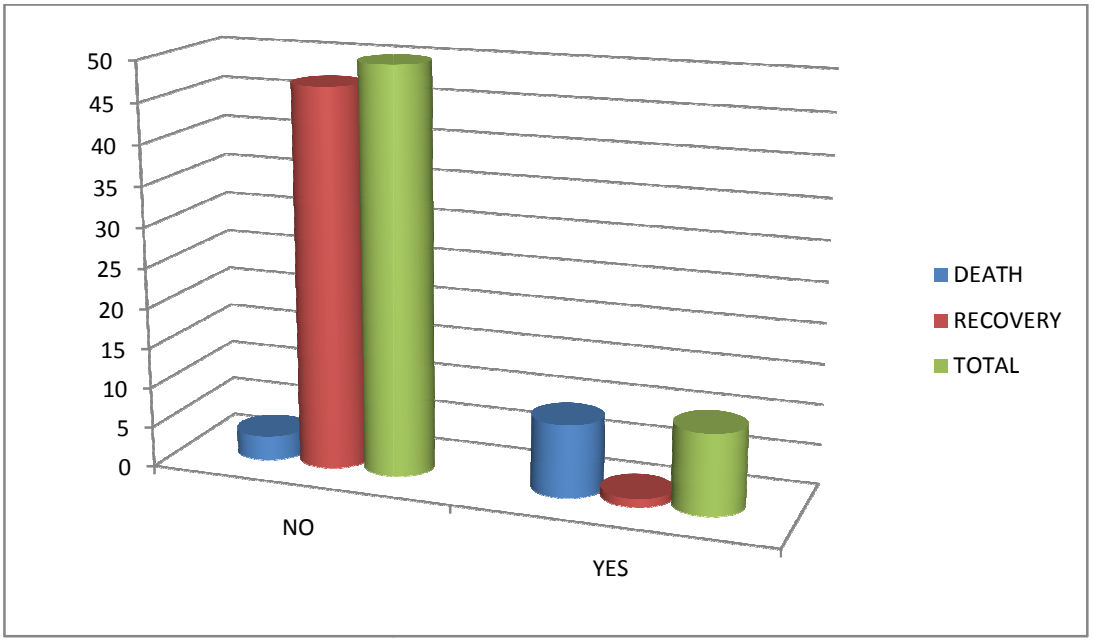


TABLE 9:

OLIGURIA	DEATH	RECOVERY	DEATH
NO	3 (25 %)	47 (97.9%)	50 (83.3%)
YES	9 (75 %)	1 (2.1%)	10 (16.7%)

FIGURE 25 : SERUM BILURUBIN MEASURED ON 4 TH DAY COMPARED WITH MORTALITY

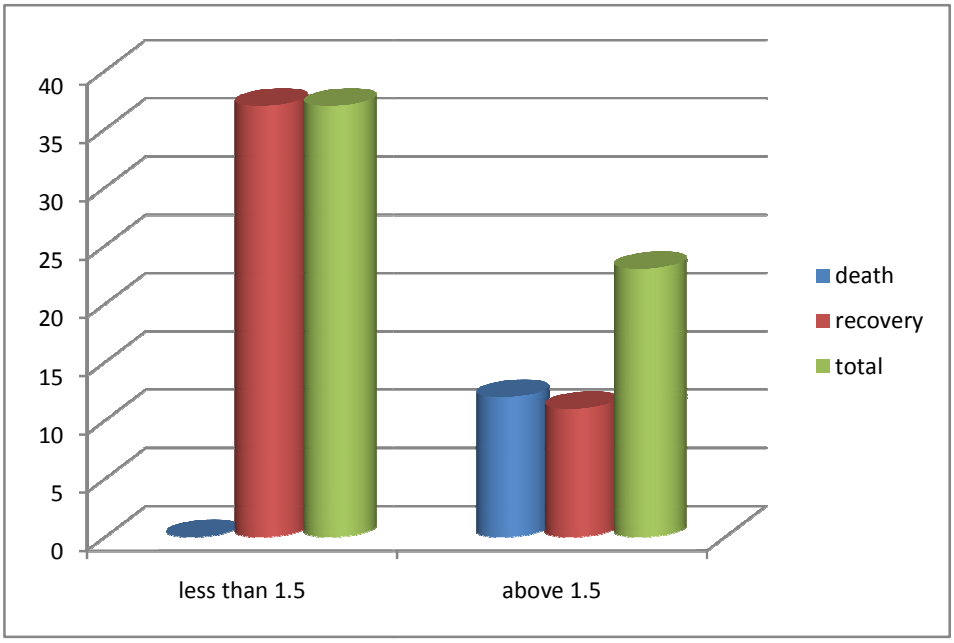


TABLE 10 :

SERUM BILIRUBIN (mg/dl)	DEATH	RECOVERY	TOTAL
LESS THAN 1.5	0 (0.0%)	37 (77.1%)	37 (61.7%)
ABOVE 1.5	12 (100.0%)	11 (22.9%)	23 (38.3%)

FIGURE 26 : SGPT LEVEL COMPARED WITH MORTALITY :

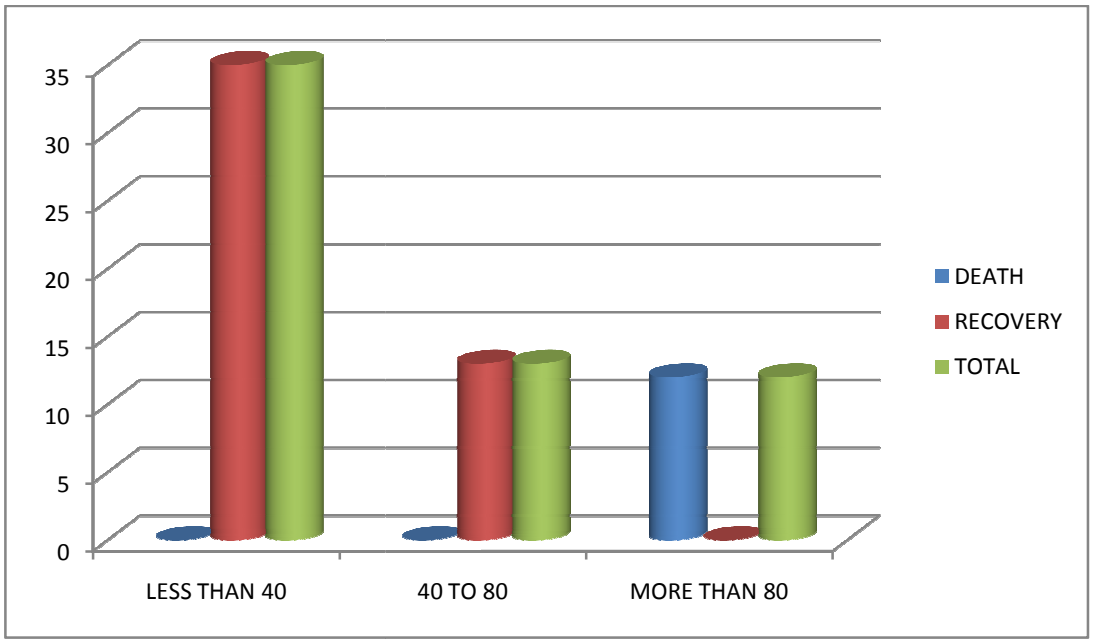


TABLE 11:

SGPT (iu/l)	DEATH	RECOVERY	TOTAL
LESS THAN 40	0 (0.0%)	36 (75 %)	36 (60 %)
41 TO 80	0 (0.0%)	11 (23.5 %)	11 (18.3%)
MORE THAN 81	12 (100.0%)	1 (1.5%)	13 (21.7%)

FIGURE 27 : SERUM CREATININE LEVEL COMPARED WITH MORTALITY :

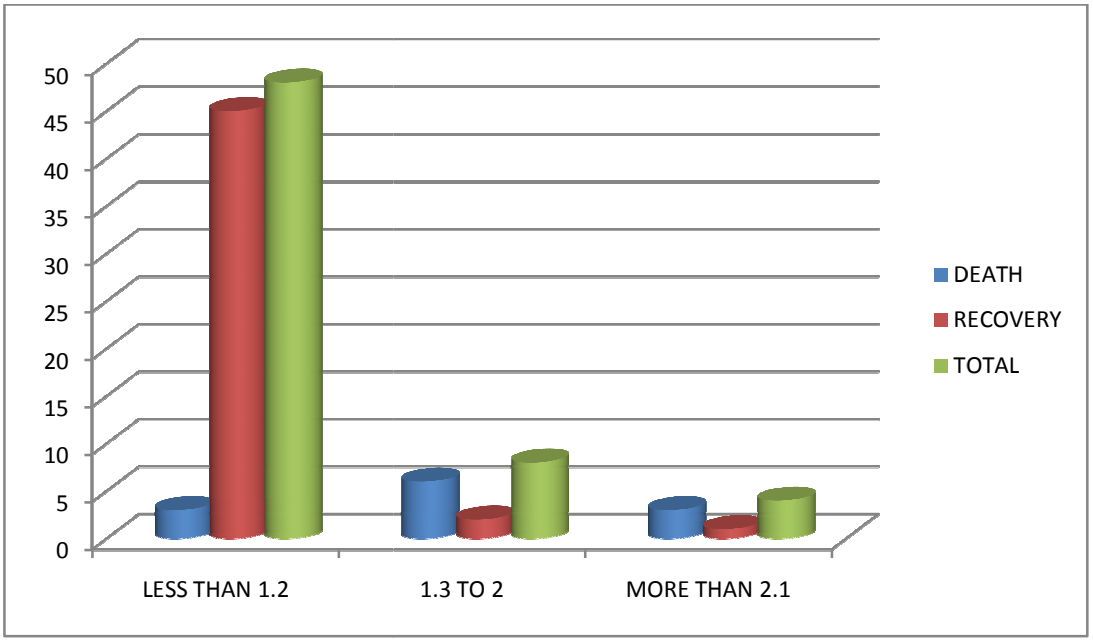


TABLE 12 :

SERUM CREATININE(mg/dl)	DEATH	RECOVERY	TOTAL
LESS THAN 1.2	3 (25 %)	45 (93.8 %)	48 (80.0%)
1.3 TO 2.0	6 (50 %)	2 (4.2 %)	8 (13.3%)
MORE THAN 2.1	3 (25 %)	1 (2.1 %)	4 (6.7%)

DISCUSSION:

This present study is a prospective longitudinal study conducted among 60 in-patients.

This present study was conducted at Thanjavur medical college from January 2012 to November 2012, total of 60 cases were studied. rodenticide being the second most common poisoning in our area , only few studies were available , hence the clinical profile of the patients were described.

Age:

In this study , most number of the patient were in the age group of 21 to 30 yrs (55%), of less than 30 yrs being 78.3%. maximum number of patients was 21 to 30 years as compared with p.s Shankar et al[55] and doshi et al[54] study.

Age	Present study (%)	Doshi et al (%)	Shankar et al(%)
< 20 yrs	23.3	-	25
21 – 30 yrs	55	44.44	60
> 31yrs	21.7	-	15

Sex:

In this study, there was male preponderance (60%) , whereas female was 40% , with male : female ratio of 1.5:1 . this corresponds to sex distribution of Shankar et al and prakash et al[55].

Sex	Present study (%)	Shankar et al (%)	Prakash et al (%)
Male	60	59.8	60
Female	40	40.2	40
Male:female	1.5:1	1.48:1	1.5:1

Marital status:-

In this study, majority of the patients were unmarried being 34 patients (56.7%), while married patients being 26 (43.3%).

Socio-economic status:-

In this study, patients from lower socioeconomic status form 78.3%, while from middle class forms 21.7%. When compared with chatterjee et al [56] and a goel et al [57] .

income	Present study (%)	Chatterjee et l(%)	A goel et al (%)
Low	78.3	88	75.73
middle	21.7	11	24.27
High	-	1	-

Quality (type) of poison ingested:-

In this study, majority of the patients – 42 (70%) were ingested paste (phosphorus compound) , next comes powder - 11(18.3%) followed by bait (super warfarins) – 7 (11.7%).

quality	Present study (%)	Vijayasekar et al (%)
Phosphorus & phosphide compounds	88.3	69
superwarfarin	11.7	31

Quantity of poison ingested:-

In this study, patient ingested less than 10 g of 35 patients (58.3%), majority being phosphorus compound . 15 to 20 g being 21 patients (35%) and more than 20 g is 4 patients (6.7%) , major being powder (phosphide

compound) . even if quantity of poison ingested is less in phosphorus compound , it produces more mortality than other compound , since it is lethal even at dose of 1 mg / kg .

Time interval between ingestion and hospitalisation:-

In this study, it was observed that mortality was higher (91.6%), who had admitted after 4 hours of ingestion, as compared to mortality in patients admitted within 4 hours, which was only meagre (8.3%). This was compared with a goel et al study.

duration	Present study		A goel et al	
	Patients(%)	Death (%)	Patients(%)	Death (%)
< 4 hrs	48.3	8.3	14.5	20
4 – 8 hrs	36.7	33.3	54.4	28.57
>8 hrs	15.0	58.3	31.1	53.12

Clinical manifestations:-

In this study , the most common symptom was abdominal pain observed in 90 % of the patients , followed by vomiting (48.3%) , next by diarrhoea (36.7%) and thirst (33.3%) . however jaundice is observed only in 13 patients (21.5%), but mortality is higher .

Biochemical analysis:-

In this study, 23 patients (38.3%- almost all ingested phosphorus compound) showed elevated serum bilirubin on 4^{th} day of more than 1.5 mg, out of which 12 patients (52.2%) had expired, which also correlate with elevated SGPT values measured on 4^{th} day; indicating the acute hepatocellular toxicity of the phosphorus compound, measuring more than five times the normal value of SGPT.

Serum creatinine was also elevated in 12 patients ingesting phosphorus compound.

Age distribution with mortality:-

In this study there was no significant difference in both incidence and mortality in age groups of the patients.

Sex distribution with mortality:-

In comparison to ingestion, however mortality in both the sex was equal of six patients each (50%), which also did not show any significant differences.

Socio-economic distribution with mortality:-

In this study, there was also no significant difference in both incidence and mortality in both lower and middle socio-economic status of the patients.

Marital status with mortality:-

In this study, there is no significant difference in mortality in both married and unmarried groups, both groups carrying equal mortality of 50 % each.

Quality of poison with mortality:-

In this study, all the mortality were among the paste group (phosphorus compound) , carrying 28.5% of mortality among ingested persons. While phosphide and warfarin group carries nil mortality.

Jaundice on 4th day with mortality:-

In this study, 13 patients (21.6%) presented with jaundice of which 12 patients (92.3%) were expired. only one patient recovered, who was been treated with N-acetyl cysteine from the day of admission, who had only survived among all the icteric patients.

Serum bilirubin on 4th day with mortality:-

In this study , 23 patients showed elevated bilirubin of more than 1.5 mg/dl , out of which 12 patients (52%) had expired , others (48%) show only mild increase , which on subsequent days decreased and patients improved.

SGPT level with mortality:-

In this study, 13 patients (28.5%) showed elevated SGPT value of more than 80 iu/ l, out of which 12 patients (92.3%) expired and one patient recovered. 11 patients (18.3%) showed values of 40 to 80 iu /l, and almost all (100%) recovered. thus SGPT value of more than five times normal along with elevated bilirubin and jaundice carries nearly full mortality.

Serum creatinine with mortality:-

In this study, 12 patients (20%) showed elevated serum creatinine values, out of which 9 patients (75%) had expired, who also had oliguria . thus it is not of much significance as other parameters in determining the mortality .

Thus out of 60 patients got admitted, 12 patients (20%) had expired, all of them had ingested paste – phosphorus compound.

Chi square test shows , there was no significant association between age , sex , socio-economic status , marital status , abdominal pain , bleeding manifestation , duration of stay and outcome of of the patient , which shows calculate value greater than table value ($p > 0.05$).

But there was significant association between quality , quantity , time delay ,thirst , diarrhoea , jaundice , oliguria , head ache , serum bilurubin on 4^{th} day , SGPT , creatinine value of the respondents and their outcomes . They show calculate value less than table value ($p < 0.05$).

Oneway ANOVA difference between SGPT, bilurubin on admn, 4th day, >1 wk, INR, creatinine of the respondents and the quality of poison

Sl.no	Quality	Mean	S.D	SS	Df	MS	Statistical inference
	on admn						
	Between Groups			2.099	2	1.049	F=6.774 .002<0.05 Significant
	Bait (n=7)	.9143	.21931				
	Paste (n=42)	1.3357	.44657				
	Powder (n=11)	.9364	.19117				
	Within Groups			8.830	57	.155	
	4th day						
	Between Groups			45.862	2	22.931	F=6.184 .004<0.05 Significant
	Bait (n=7)	.9429	.26992				
	Paste (n=42)	2.5310	2.25318				
	Powder (n=11)	.4545	.52795				
	Within Groups			211.374	57	3.708	
	>1 wk						
	Between			37.338	2	18.669	F=3.491

	Groups						.037<0.05 Significant
	Bait (n=7)	.0000	.00000				
	Paste (n=42)	1.7214	2.72661				
	Powder (n=11)	.0000	.00000				
	Within Groups			304.811	57	5.348	
	INR						
	Between Groups			4.164	2	2.082	F=5.732 .005<0.05 Significant
	Bait (n=7)	2.1914	.55364				
	Paste (n=42)	1.5493	.67715				
	Powder (n=11)	1.2073	.08063				
	Within Groups			20.704	57	.363	
	SGPT						
	Between Groups			47.111	2	29.055	F=6.050 0.04<0.05 Significant
	Bait (n=7)	0.9129	0.35336				
	Paste (n=42)	2.6190	2.14607				
	Powder (n=11)	0.4545	0.5323				
	Within Groups			213.489	57	3.623	

	creatinine						
	Between Groups			1.469	2	.734	F=2.609 .082<0.05 Significant
	Bait (n=7)	.9429	.69488				
	Paste (n=42)	1.1286	.55887				
	Powder (n=11)	.7273	.18488				
	Within Groups			16.045	57	.281	

Statistical test: *Oneway ANOVA 'f' test was used the above table*

Inference:

The above table indicates that there is a significant difference between bilurubin on admn, 4th day, >1 wk, sgot, INR, creatinine of the respondents and their quality. Hence the calculate value less than table value *(p<0.05)*.

CONCLUSION:-

1) Rodenticide poisoning is the second most common poisoning in our area.

2) Male to female ratio in our study is 1.5: 1.

3) Majority of the patients were in 20 to 30 years age group.

4) Many of them are from low socioeconomic status.

5) Phosphorus compound (paste) forms the major share of poison.

6) Most common symptom is abdominal pain (90%).

7) Bleeding manifestation is common in warfarin group (bait).

8) Mortality in this study was 20 %.

9) Increased time delay in hospitalisation carries more mortality.

10) Mortality is seen only in phosphorus compound.

11) Jaundice develop following ingestion of phosphorus, carries most mortality, which also correlates with elevated serum bilirubin and SGPT level.

12) N-acetyl cysteine can be tried in early stages of hepatotoxicity .

13) Phosphorus (paste) compound of rodenticide , must be banned , to prevent mortality in young productive poor patients , who intend to ingest poison in a minute decision .

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ANNEXURE I:

ABBREVATIONS :-

1. ALT : ALanine Transaminase (or) ALanine Aminotransferase.
2. ANTU : Alpha – Naphthyl Thio Urea.
3. DIC : Disseminated Intravascular Coagulation.
4. ECG : Electro CardioGram.
5. FFP : Fresh Frozen Plasma.
6. GIT : Gastro Intestinal Tract.
7. INR : Internatinational Normalized Ratio.
8. LD 50 : Lethal Dose in 50% of individuals.
9. ppm : parts per million.
10. PT : Prothrombin Time.
11. SGPT : Serum Glutamic Pyruvic Transaminase.

PROFORMA

NAME :

AGE :

SEX :

ADDRESS :

IP No :

DOA :

SOCIO-ECONOMIC STATUS : lower/middle/upper

HISTORY :

Informer : patient/ relative

Quality of the compound : bait/paste/powder

Quantity consumed : g

Whether taken with alcohol : yes/no

Time interval from ingestion to hospitalisation : hrs

Cause of poisoning : suicidal / accidental

Associated evidences

Container :

Specific colour of vomitus :

Gastric lavage : colour / smell

Staining in body parts or dress:

Symptoms presented:

1) Vomiting :
2) Abdominal pain :

3) Loose stools :
4) Yellow coloured secretions :
5) Decreased urine output :
6) Bleeding tendencies :
7) CNS : headache/ seizures/coma

Past history:

Consumed poison before : yes/no

Any serious illness : yes/no

Known psychiatric illness : yes/no

Jaundice earlier : yes/no

Known liver disease : yes/no

Known alcoholic : yes/no

Treated and referred from other hospitals:yes /no

GENERAL EXAMINATION :

Conscious :

Icterus :

Vitals pulse :

BP :

SYSTEMIC EXAMINATION :

CVS :

RS :

ABDOMEN :

CNS :

LAB INVESTIGATIONS

RBS :

UREA :

SERUM CREATININE :

SERUM ELECTROLYTES :

LIVER FUNCTION TESTS

Bilirubin Total :

Indirect :

Direct :

SGOT :

SGPT :

ALP :

Protein :

PT :

INR :

OUTCOME :

.

ANNEXUREIII:

KEY TO MASTER CHART :

1) s.no : serial number

2) i.p no **:** inpatient number.

3) On admn : on admission.

4) 4^{th} day : on fourth day.

5) > 1 wk : more than one week.

6) INR : International Normalised Ratio.

7) Sgpt : serum glutamic pyruvic transaminase .

8) R : Recovery.

9) D : Death .

10) m **:** male .

11) f : female .

12) l : low socio-economic status.

13) m : middle socio-economic status.

14) s : single.

15) m : married .

16) 0 : no symptoms.

17) 1 : presence of symptoms.

ANNEXURE II :

KEY TO PROFORMA :

1) I p no : inpatient number .

2) DOA : Date Of Admission .

3) CNS : Central Nervous System .

4) BP : Blood Pressure .

5) CVS : Cardio Vascular System .

6) RS : Respiratory System .

7) RBS : Random Blood Sugar .

8) SGOT : Serum Glutamic ornithyl Transferase.

9) SGPT : Serum Glutamic Pyruvic Transamnase

10) PT : Prothrombin Time.

11) INR : International Normalised Ratio .

s.no	name	age	sex	i.p no	socioeconomic status	marital status	quality	quantity	time delay	syr			
										vomiting	abdominal pain	thirst	diarrhoea
1	senthil kumar	24	m	1372275	l	s	powder	20 g	< 4 hrs	0	1	0	0
2	ramya	17	f	1373382	l	s	paste	5 g	< 4 hrs	o	1	0	0
3	boopathy	20	m	1374970	m	s	bait	5 g	< 4 hrs	0	1	0	0
4	prakash	28	m	1377082	m	m	paste	20 g	4-8 hrs	1	1	0	1
5	loganathan	21	m	1377918	l	s	paste	10 g	4-8 hrs	1	1	0	1
6	ajith kumar	14	m	1378408	l	s	powder	15 g	< 4 hrs	1	0	0	0
7	muthukumaran	30	m	1378861	m	m	paste	5 g	< 4 hrs	0	1	0	1
8	annamalai	22	m	1379467	l	s	paste	10 g	> 8 hrs	1	1	1	1
9	joseph	58	m	1379572	l	m	bait	10 g	4-8 hrs	0	1	1	0
10	rasiya	30	f	1382582	l	m	paste	15 g	< 4 hrs	0	1	0	1
11	sagayaraj	27	m	1381984	l	m	paste	5 g	4-8 hrs	1	1	0	0
12	raja	24	m	1382838	l	s	paste	10 g	< 4 hrs	1	1	0	1
13	sivalingam	24	m	1383406	l	s	paste	15 g	> 8 hrs	1	1	1	1
14	karthika	21	f	1383916	m	s	powder	25 g	< 4 hrs	0	1	0	1
15	vadivel	55	m	1385406	l	m	paste	5 g	< 4 hrs	1	1	0	0
16	muthumani	13	f	1387759	l	s	bait	5 g	< 4 hrs	0	0	0	0
17	rajalakshmi	25	f	1388085	l	m	paste	20 g	4-8 hrs	1	1	1	1
18	gunasekaran	26	m	1389455	l	s	paste	10 g	< 4 hrs	0	1	0	1
19	sakuntala	50	f	1389706	l	m	paste	20 g	> 8 hrs	1	1	1	0
20	sambandam	36	m	1391199	m	m	paste	15 g	4-8 hrs	0	1	1	0
21	prabhudeva	18	m	1392061	l	s	paste	5 g	< 4 hrs	1	1	0	0
22	rengasamy	75	m	1392544	l	m	paste	5 g	< 4 hrs	0	1	0	0
23	deivakani	25	f	1392557	l	m	paste	5 g	< 4 hrs	1	1	0	0

s.no	name	age	sex	i.p no	socioeconomic status	marital status	quality	quantity	time delay	syr			
										vomiting	abdominal pain	thirst	diarrhoea
24	aravindraj	23	m	1392606	l	s	powder	25 g	4-8 hrs	0	0	0	0
25	sakuntala	32	f	1393188	l	m	paste	15 g	> 8 hrs	1	1	1	1
26	karthik	23	m	1395065	l	s	bait	5 g	4-8 hrs	0	1	1	0
27	kumaresan	19	m	1395852	l	s	paste	10 g	< 4 hrs	0	1	0	0
28	mansur ali	18	m	1395916	m	s	powder	20 g	> 8 hrs	0	1	0	0
29	kamala	39	f	1396530	l	m	paste	15 g	< 4 hrs	0	1	1	1
30	vasuki	35	f	1398279	l	m	paste	5 g	4-8 hrs	0	1	1	0
31	sathyaraj	23	m	1398455	m	s	paste	15 g	4-8 hrs	1	1	1	0
32	paramanandham	28	m	1398513	l	m	powder	25 g	< 4 hrs	0	1	1	0
33	ragavan	55	m	1398580	m	m	paste	5 g	< 4 hrs	0	1	1	0
34	dinesh kumar	24	m	1398638	l	s	powder	20 g	4-8 hrs	0	1	0	0
35	shakila	17	f	1399142	l	s	paste	5 g	< 4 hrs	0	1	0	1
36	karnan	40	m	1399461	l	m	paste	10 g	4-8 hrs	1	1	0	0
37	manibharathi	21	m	1400006	l	s	paste	5 g	4-8 hrs	1	1	1	1
38	nathiya	30	f	1400079	l	m	paste	10 g	4-8 hrs	1	1	0	0
39	saranya	20	f	1401321	m	s	bait	5 g	< 4 hrs	0	1	1	0
40	sivakumar	14	m	1402557	l	s	paste	10 g	4-8 hrs	1	1	0	1
41	indirani	35	f	1403446	l	m	powder	30 g	< 4 hrs	1	0	0	0
42	mahalakshmi	23	f	1403692	l	m	paste	5 g	4-8 hrs	0	1	1	0
43	revathi	20	f	1404255	l	s	paste	15 g	> 8 hrs	1	1	1	0
44	sathya	27	f	1404690	m	m	paste	10 g	> 8 hrs	0	1	0	1
45	amuthan	30	m	1404975	l	s	paste	10 g	4-8 hrs	1	1	0	0
46	abdullah	20	m	1405889	m	s	paste	10 g	< 4 hrs	1	1	0	0

s.no	name	age	sex	i.p no	socioeconomic status	marital status	quality	quantity	time delay	syr			
										vomiting	abdominal pain	thirst	diarrhoea
47	malathi	30	f	1407239	l	m	powder	20 g	4-8 hrs	0	0	0	0
48	madhavan	26	m	1407782	l	s	paste	10 g	< 4 hrs	0	1	1	1
49	venkatesan	29	m	1408901	l	s	paste	20 g	> 8 hrs	1	1	1	1
50	mangayarkarasi	27	f	1408945	l	m	paste	15 g	> 8 hrs	1	1	0	1
51	vinotha	22	f	1409261	l	m	bait	10 g	< 4 hrs	0	1	0	0
52	subashini	24	f	1409397	m	s	paste	15 g	< 4 hrs	1	1	0	0
53	durgadevi	22	f	1410404	l	s	powder	20 g	4-8 hrs	1	1	0	0
54	valli	35	f	1412127	l	m	paste	10 g	4-8 hrs	0	1	0	1
55	siva	30	m	1412707	l	s	paste	15 g	< 4 hrs	1	1	0	0
56	murugesan	40	m	1413384	l	m	paste	10 g	< 4 hrs	0	1	0	1
57	kannan	26	m	1413820	l	s	powder	15 g	< 4 hrs	0	0	0	0
58	tamil ilakiya	17	f	1414549	m	s	bait	5 g	4-8 hrs	1	1	0	0
59	ramesh	20	m	1415791	l	s	paste	5 g	< 4 hrs	1	1	0	0
60	gopu	26	m	1416245	l	s	paste	10 g	4-8 hrs	0	1	1	1

s.no	name	mptoms				serum bilurubin			INR	sgpt	creatinine	Duration of stayal	outcome
		jaundice	bleeding manifestations	oliguria	headache	on admn	4th day	>1 wk					
1	senthil kumar	0	0	0	0	1.2	_	_	1.1	28	0.8	3 days	R
2	ramya	0	0	0	0	1.1	1	_	1.84	27	0.7	5 days	R
3	boopathy	0	1	0	0	0.8	1	_	1.3	42	0.6	7 days	R
4	prakash	1	0	1	0	1.4	3.2	7.6	2.3	344	2.4	8 days	D
5	loganathan	0	0	0	0	0.9	1.6	1.1	1.22	44	0.8	9 days	R
6	ajith kumar	0	0	0	0	0.7	1.1	_	1.1	34	0.8	3 days	R
7	muthukumaran	0	0	0	0	1.4	1	_	1.46	26	0.6	6 days	R
8	annamalai	1	0	1	1	1.6	3.8	6.3	1.3	278	2.5	7 days	D
9	joseph	0	1	0	0	1.2	1.4	_	2.34	29	0.7	5 days	R
10	rasiya	0	0	0	0	1.o	2.1	1.3	1.26	37	1.2	6 days	R
11	sagayaraj	0	0	0	0	0.9	1.7	1.3	1.2	34	0.9	8 days	R
12	raja	0	0	0	0	1.3	1.1	1.1	1.32	42	1	7 days	R
13	sivalingam	1	0	1	1	3.4	12.4	_	1.9	463	2	6 days	D
14	karthika	0	0	0	0	0.9	1	_	1.16	26	0.7	4 days	R
15	vadivel	0	0	0	0	1.6	1.3	_	1.24	51	1.4	5 days	R
16	muthumani	0	0	0	0	0.7	0.6	_	1.7	38	0.6	4 days	R
17	rajalakshmi	1	0	1	0	1.7	4.8	_	1.6	410	1.9	4 days	D
18	gunasekaran	0	0	0	0	1.2	1.7	1.6	1.3	52	0.9	8 days	R
19	sakuntala	1	0	0	1	1.6	5.8	_	1.9	374	1.8	4 days	D
20	sambandam	0	0	1	0	1	1.9	1.3	1.2	47	1	9 days	R
21	prabhudeva	0	0	0	0	1.2	0.8	_	1.16	43	0.9	5 days	R
22	rengasamy	0	0	0	0	1.2	1	_	1.1	24	1.2	5 days	R
23	deivakani	0	0	0	0	1.2	1.4	_	1.26	54	0.7	6 days	R

s.no	name	mptoms				serum bilurubin			INR	sgpt	creatinine	Duration of stayal	outcome
		jaundice	bleeding manifestations	oliguria	headache	on admn	4th day	>1 wk					
24	aravindraj	0	0	0	0	0.9	_	_	1.24	26	0.6	3 days	R
25	sakuntala	1	0	1	0	0.8	5.8	_	2.8	285	2.7	5 days	D
26	karthik	0	1	0	0	0.7	0.8	_	2.3	32	0.6	4 days	R
27	kumaresan	0	0	0	0	0.8	0.9	_	1.2	22	0.7	6 days	R
28	mansur ali	0	0	0	0	0.8	0.8	_	1.2	26	0.5	5 days	R
29	kamala	0	0	0	0	1.4	3	5.3	2.5	352	1.8	7 days	D
30	vasuki	0	0	0	0	1.4	1.2	_	1.2	39	0.9	6 days	R
31	sathyaraj	1	0	1	0	1.2	4	6.2	1.3	242	1.9	8 days	D
32	paramanandham	0	0	0	0	0.8	_	_	1.12	37	0.7	3 days	R
33	ragavan	0	0	0	0	1.4	1.2	_	1.2	42	0.9	6 days	R
34	dinesh kumar	0	0	0	0	0.7	_	_	1.3	32	0.7	3 days	R
35	shakila	0	0	0	0	0.9	1.2	1	1.2	34	0.6	6 days	R
36	karnan	0	0	0	0	1.3	1.8	1.4	1.1	46	1.2	7 days	R
37	manibharathi	0	0	0	0	1.2	1.5	1.1	1.2	26	0.7	7 days	R
38	nathiya	0	0	0	0	1.6	1.4	_	1.3	39	1	6 days	R
39	saranya	0	0	0	0	1.2	1.1	_	2.7	42	0.7	4 days	R
40	sivakumar	1	1	0	0	1.2	3.7	6.4	1.9	176	0.9	7 days	D
41	indirani	0	0	0	0	1	_	_	1.2	32	0.6	2 days	R
42	mahalakshmi	0	0	0	0	1.2	1	_	1.14	24	0.6	5 days	R
43	revathi	1	1	1	0	2.3	6.8	12	4.8	157	0.6	9 days	D
44	sathya	0	0	0	0	0.8	1.6	1.2	1.4	36	0.7	8 days	R
45	amuthan	0	0	0	0	1.6	1.3	_	1.3	31	0.6	5 days	R
46	abdullah	1	0	0	0	1.6	1.3	1	1.32	26	1.3	7 days	R

s.no	name	mptoms				serum bilurubin			INR	sgpt	creatinine	Duration of stayal	outcome
		jaundice	bleeding manifestations	oliguria	headache	on admn	4th day	>1 wk					
47	malathi	0	0	0	0	1.2	_	_	1.24	25	1.2	3 days	R
48	madhavan	0	0	0	0	1.6	1.8	1.3	1.26	29	0.9	7 days	R
49	venkatesan	1	0	1	1	1.4	5.8	_	1.6	374	2.3	4 days	D
50	mangayarkarasi	1	1	1	0	1.2	5.2	7.3	2.8	317	2	6 days	D
51	vinotha	0	1	0	0	1	1	_	2.1	28	0.7	4 days	R
52	subashini	0	0	0	0	1.7	2.3	1.6	1.9	37	0.9	18 days	R
53	durgadevi	0	0	0	0	1.2	1.1	_	1.3	26	0.6	4 days	R
54	valli	0	0	0	0	1.3	1.7	1.4	1.24	36	0.7	8 days	R
55	siva	0	0	0	0	1.4	1.6	1.4	1.33	28	0.9	6 days	R
56	murugesan	0	0	0	0	1.2	1.4	1.1	1.25	29	1	6 days	R
57	kannan	0	0	0	0	0.9	1	_	1.32	32	0.8	4 days	R
58	tamil ilakiya	0	0	0	0	0.8	0.7	_	2.9	24	0.9	4 days	R
59	ramesh	0	0	0	0	0.9	1.3	1	1.15	42	0.8	6 days	R
60	gopu	0	0	0	0	1	0.9	_	1.12	34	0.7	5 days	R

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College	:	THANJAVUR MEDICAL COLLEGE
Dissertation Topic	:	CLINICAL PROFILE OF PATIENTS ADMITTED WITH RODENTICIDE POISONING

The Ethical Committee, Thanjavur Medical College has decided to inform that your Dissertation Topic is accepted and you are permitted to proceed with the above study.

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